A TREATISE

ON COMMON FORMS OF

FUNCTIONAL NERVOUS DISEASES

BY

L. PUTZEL, M.D.,

Physician to the Clinic for Nervous Diseases, Bellevue Hospital Out-Door Department; Visiting Physician for Nervous Diseases, Randall's Island Hospital; Pathologist to the Lunatic Asylum, B. I.; Curator to Charity Hospital, etc.

NEW YORK:
WILLIAM WOOD & COMPANY,
27 GREAT JONES STREET.
1880.

PREFACE.

PATHOLOGICAL anatomy has exercised such an enormous influence upon the advances made in practical medicine within the last twenty-five years that many pathologists sneer at the term "functional" disease and deny its very existence.

While we fully agree that there can be no morbid manifestations without a change in the material structure of the organs involved, we are nevertheless fully convinced, in view of the fruitless search of pathological anatomists, that the diseases which we have considered in this work present no primary anatomical changes which are visible to the naked eye or to the microscope—in other words, that the changes in structure are of a molecular nature.

Perhaps in the reviving growth of physiological chemistry, the balance of the chemist will determine the presence of subtle changes in the constitution of the diseased organs, and will afford us some insight into the true pathogeny of these affections.

The tendency to the disbelief in the actual existence of functional nervous diseases led to the inconvenience that their clinical study has been neglected.

If we glance through the most widely known text-books on nervous diseases which have appeared in the English language, we will find that due attention is not paid to functional affections, although practically they are by far the most important, and are much more frequently encountered by physicians than diseases due to organic lesions. A change is now, however, becoming noticeable in this respect, especially in foreign literature.

In the present work special attention has been paid to the sections on clinical history and diagnosis, as it is especially in the latter respect that mistakes are made. I have entered into the pathology of the affections merely with the view of giving the present status of our actual knowledge of the subject, and not for the discussion of disputed questions.

The consideration of hysteria has been omitted because this dis-

ease has been described in sufficient detail in numerous works which are now in the hands of the medical public. It is true that the French school have recently revealed some startling and hitherto undescribed manifestations of hysteria, which they have included under the term hystero-epilepsy, and which have excited a great deal of interest in scientific circles. In our own country, however, these symptoms are observed so rarely that I have not considered myself justified in entering into the subject in a work of this character.

Some of the forms of peripheral paralysis which have been considered in the final article cannot be regarded, properly speaking, as functional, but I have discussed them under that heading in accordance with long-established custom and for the sake of completeness.

In conclusion, I desire to acknowledge my great indebtedness to my friends Drs. V. P. Gibney, R. Van Santvoord, and S. Hemingway, for much valuable assistance received in the preparation of this work.

L. PUTZEL.

252 EAST FORTY-EIGHTH STREET,
NEW YORK, July 20, 1880.

CHOREA.

CHAPTER I.

CLINICAL HISTORY.

CHOREA is essentially a disease of childhood, though it occasionally begins in adult life, and, in rare instances, makes its appearance in old age. Its first beginnings in children are frequently misinterpreted as the results of wilfulness. A child, while attending school, is noticed to become restless and frequently move from side to side in his seat. Twitchings of the muscles of the hands also develop and are evidenced by irregularity in writing, in the manner of holding a pen or pencil, and by the fact that the patient will drop his slate or some other light object more frequently than his fellows. In addition, certain slight manifestations of mental disorder make their appearance at the same time as the development of the muscular phenomena, or may even precede the latter. These symptoms consist of slight loss of memory, and inability of the patients to apply themselves to their studies as well and continuously as formerly. Children who were previously of an obedient and mild disposition become irritable, obstinate and perverse. They become insubordinate, lose their love of play, and are not so affectionate as was their wont. These phenomena are naturally looked upon as indubitable evidences of wilfulness and are punished accordingly, thus frequently precipitating and aggravating the course of the disease. The muscular symptoms gradually spread in intensity and extent, and usually attain their maximum severity in the course of a couple of weeks to a month. At the height of the disease, all the voluntary muscles in the body, especially those of the arms and face, may be affected by the choreic movements. The eyelids twitch irregularly, the ocular muscles proper undergo similar involuntary contractions, and those inserted into the angles of the mouth twitch in an irregular manner, imparting to the face a peculiar expression, sometimes bordering on the ludicrous, sometimes of a stern character. At times the mouth is thrown widely open and the tongue is rapidly protruded, to be as quickly withdrawn. The muscles of the neck, especially the sterno-cleido-mastoids, are also involved, causing the head to bend rapidly upon the chest, to be thrown backward, or to be tossed from side to side. In rare instances, the affection is confined to the muscles of the neck, and the head is continually engaged in a series of nodding movements, constituting the so-called *chorea nutans* of Marshall Hall. It is doubtful, however, whether this is a form of true chorea. The muscles of respiration and phonation are not exempt from the convulsive phe-

nomena. If the chest is bared, it will be found that inspiration is frequently interrupted, before the act is complete, by a sudden expiratory movement. The speech of the patient is often interrupted, owing to the irregularity in the respiratory rhythm. This disturbance is sometimes so marked that the patients must take a deep inspiration between the utterance of individual words, and become greatly fatigued after speaking a few sentences. According to Ziemssen,[1] an affection of the laryngeal muscles usually accompanies severe cases of chorea. By means of the laryngoscope, Ziemssen was able to detect the irregular contractions of the different muscles which are engaged in varying the tension of the vocal cords. In some cases, this is shown by the *low pitch* and *monotony* of the voice; as improvement progresses, these phenomena gradually disappear. In many instances the hands are chiefly affected. Even when the child is perfectly quiescent and without any voluntary effort on his part, the hands will suddenly become forcibly flexed or extended, the fingers will be separated from one another, and the hand will be drawn away from the body, or *vice versa*. If the patient attempts to drink a glass of water, he experiences great difficulty in grasping the glass, and is unable to carry it directly to his mouth. A considerable portion of its contents is spilled in the endeavor to drink, and the patient may strike the vessel against his cheek or nose. The legs are also usually affected, though not, as a rule, to such an extreme degree as the hands. In walking, the knees frequently bend under, the patient trips and stumbles on account of the ill-timed contractions of the extensors or flexors of the foot, and the legs often become entangled in one another; sometimes, however, a shuffling gait is the only evidence of the affection of the legs. In severe forms of the disease, the patient is in a truly deplorable condition. The movements of the hands may be so intense that he is unable to feed himself, and when the muscles of mastication and deglutition are implicated to an extreme degree, the sufferer must be fed through the stomach-tube or by means of nutritive enemata. Sometimes the patient is unable to maintain his seat, but is continually jumping up and down or falling from his chair. Even in cases of moderate severity, locomotion is often rendered difficult, if not impossible, by the irregular contractions occurring in the legs. As a rule, the movements cease almost entirely during sleep (although rest is often disturbed by frequent fits and starts); but, in cases of extreme severity, sleep is rendered impossible by the violence of the movements, and the latter continue even when the patient does finally fall into a light slumber. Ulcerations of the prominent portions of the body, from the continued friction against surrounding objects, supervene in these cases, and inanition and exhaustion rapidly develop from the lack of nutrition consequent on the inability to eat, from the continuous violence of the choreiform movements, and from the loss of sleep.

In light cases, the choreic contractions usually occur when the patient endeavors to perform a voluntary act, and the bizarre movements are then due to associated contractions of antagonistic muscles. In the more severe forms, the movements develop irrespective of any voluntary effort on the part of the patient, and, as we have previously mentioned, may even continue uninterruptedly during sleep. But these statements will not hold good in all cases and under all circumstances. Gowers [2]

[1] Handb. d. spec. Path. u. Therap. Bd. XII. p. 415.
[2] Brit. Med. Journ., Mar. 30, 1878.

found, that, in chorea, inco-ordination of voluntary movement bears no relation to the spontaneous movements, but varies independently of the latter. Thus, a patient in whom the spontaneous movements were very marked, could perform voluntary actions with great ease and steadiness, while, on the other hand, in a patient with scarcely noticeable spontaneous movements, the inco-ordination became extreme when he tried to execute a voluntary effort. The independent variation in these two elements in chorea suggests that they may depend on an affection of distinct and separate regions of the nerve-centres.

This state of affairs appears, however, to be decidedly exceptional. The majority of authors do not appear to have had a similar experience, and I do not recollect a single case in which the statements of Gowers will hold good, although I have carefully examined in this particular all the patients who have come under my observation since reading Gowers's article.

The movements are not always bilateral, but may only involve one half of the body, and then constitute a variety of the affection usually known as hemichorea. The disease not infrequently begins as a hemichorea, but spreads to the other side of the body as the affection progresses. The following statistics will serve to show the relative frequency of this variety of the disease:

	No. of cases.	Hemichorea.
Sée	154	97
Pye-Smith	150	33
Russell	97	29
Author	82	16

Among Pye-Smith's 33 cases of hemichorea, 15 affected the right and 18 the left side of the body. Althaus reported 22 cases of hemichorea, of which 16 involved the left and 6 the right side of the body; Ogle, 24 right, 20 left hemichorea; Russell, 18 right, 11 left. These facts possess considerable importance and we shall refer to them hereafter in discussing the pathology of this affection. In rare instances, the choreiform movements are even more limited in their distribution and may be confined to the face or arm. In one case of vertebral caries in the lower lumbar region, which was under my observation, the patient, a boy æt. 16 years, began to suffer from well developed choreic movements of the left forearm and hand, and the left side of the face, these phenomena suddenly making their appearance upon the day after he had seen a severe case of general chorea in a young friend.

It is a curious and interesting fact that the patients do not complain much of fatigue, even although the movements are quite violent and continuous. As a rule, also, there is little or no loss of power in the affected limbs, though in some cases considerable paresis may develop, especially in hemichorea. The intensity of the paresis does not appear to present any definite relation to the severity of the choreiform movements. A better idea of the loss of power can be obtained by allowing the patient to squeeze the hand of the observer than by using the dynamometer, since the manipulation of the latter requires more delicate muscular co-ordination than the former procedure, and is therefore more interfered with by the choreic movements. In rare instances the muscles become completely paralyzed. I shall report in full the following case of general paralysis due to chorea, as it is very interesting from several points of view.

CASE I.—The patient, Peter K., æt. 3¾ years, first came under my observation at the clinic for Nervous Diseases in the Bellevue Out-door Department, on April 22, 1878. The family history is unimportant; neither of the parents or other members of the family have ever suffered from rheumatism. The patient was always in excellent health until seven weeks previously, when he suddenly developed considerable fever, followed in three days by swelling of the knees and ankles, the joints also becoming exceedingly painful to the touch. This condition lasted three weeks, and was diagnosed as acute articular rheumatism by the physician in attendance. Very shortly after the termination of the rheumatic attack (about April 1st), and while the patient was apparently doing very well, he began to suffer from irregular choreiform twitchings in the limbs, which did not, however, attain any considerable severity. A few days after this symptom appeared, the child awoke one morning in a condition of great muscular weakness, and was unable to articulate, although speech had hitherto been perfect. The paresis gradually grew worse until a week ago (April 15th), since which time it has remained *in statu quo*. The twitchings of the muscles continued up to the present, but were not very marked.

Present condition (April 22, 1878).—The patient is a large child, apparently well nourished. He has left convergent strabismus, which came on after a slight attack of diarrhœa, that occurred last July, and has persisted ever since. Physical examination: lungs normal; the apex-beat of the heart is felt at the nipple; a loud, blowing systolic murmur is present, which is heard most distinctly at the apex and is also conveyed into the left axillary space, but could not be traced into the scapular region; the second cardiac sound is heard sharply and distinctly, and is not accompanied by any adventitious sounds.

The patient is unable to swallow solid food, and this condition has lasted since the beginning of the paralysis. There is considerable weakness of the upper limbs; the grasp is feeble, and the patient is unable to raise his hands above the shoulders. The lower limbs are even weaker than the upper. There is almost complete paralysis of the anterior muscles of the legs, the feet hanging in the position of talipes equinus, and the toes are only movable to a very slight extent. The patient is barely able to flex the thighs on the abdomen. When placed in a sitting posture the child immediately topples over to one or the other side, apparently from paralysis of the dorsal muscles. The muscles throughout the entire body feel soft and flabby. It is impossible to get accurate data with regard to sensation, on account of the age of the patient; reflex action and the electro-muscular reactions are normal throughout the body. The patient is unable to speak, except to say "yes" and "no," the former word being uttered in a very indistinct manner. Sleep is very much disturbed. The natural folds of the face are almost completely effaced and the facial muscles appear to be paretic, if not entirely paralyzed; the features present a dull mask-like appearance. The patient is, however, able to close the eyes in a normal manner. There is very little power of motion in the tongue, the organ being protruded very slowly and tremulously, and only to a slight extent. The choreiform movements are scarcely noticeable, come on only at long intervals, and are very moderate in intensity. I ordered milk diet, ol. morrhuæ, and a mixture composed of tinct. ferri chlorid. ʒ ij., potass. chlorat. ʒ j., and syr. simp. ℥ iv., one teaspoonful being given three times a day. The child began to mend very rapidly, and on May 22d the following notes were taken: the patient is

able to walk almost as well as ever, and the power in the upper extremities is apparently entirely restored; slight choreiform movements still continue from time to time. Speech is almost perfect. The mother states that during the last two weeks the child has been almost demented, and has not appeared to comprehend the simplest ideas. At times he has apparently had hallucinations of sight. On one occasion, while playing with some toys on a sofa, he began to talk with imaginary playmates, saying "that he was better than they." At other times, he is said to have had hallucinations of various kinds. To-day, however, the patient appears quite bright and intelligent.

June 5th.—The patient was again brought to me and was entirely well in every particular, except that the heart-murmur was still distinctly audible, though not so loud as formerly.

This case is unlike those in which the muscles become paretic during the course of very intense choreic movements; in fact, the latter were so slight at times that prolonged observation was necessary in order to detect them.

Clifford Albutt [1] also published the notes of an attack of acute chorea followed by a state of general paralysis.

C. Handfield Jones [2] narrates the case of a girl suffering from chorea, attended with extreme paresis of the limbs and trunk, and also of the tongue and pharynx; no rheumatism or heart-murmur discoverable. The patient recovered completely. The cases in which an attack of chorea precedes hemiplegia (præhemiplegic chorea), and those in which it follows the latter (post-hemiplegic chorea), together with the allied affection known as athetosis, will be discussed in a subsequent section of this article. Trousseau, in his "Lectures on Clinical Medicine," mentions the case of a girl æt. 18 years, who, after an attack of right hemiplegia, manifested symptoms of hemichorea upon the paralyzed side. Trousseau regarded this case as one of ordinary chorea, but there is very little doubt that it belongs to the category of post-hemiplegic chorea, which is of an entirely different nature, and the pathological significance of which was unknown at the period during which this observation was made.

Very few investigations have been made with regard to the electrical reactions of the nerves and muscles in chorea. Rosenthal [3] resorted to electrical exploration in three cases of hemichorea observed soon after the onset of the disease (for obvious reasons it is useless to make examinations of this nature in cases in which the choreic movements occur upon both sides of the body). He found a marked increase of electro-muscular contractility in the affected muscles. Rosenthal observed very marked excitability to the galvanic current, which was manifested by contractions upon closure at the negative pole, by galvano-tonic contractions with weak currents, and by contractions upon opening at the cathode. Increased irritability of the sensory nerves was also manifested. Gowers [4] states that a few weeks after the onset of the affection there is observed in most, though not in all cases, a distinct increase in the irritability of the nerves and muscles of the affected side, both to the faradic and the galvanic currents. The difference varied from 1—2 centimetres of the

[1] Medical Times and Gazette for 1878.
[2] Practitioner, vol. xxi., 1878.
[3] A Clinical Treatise on Diseases of the Nervous System, 1878.
[4] Brit. Med. Journ., March 30, 1878.

secondary coil of Stoehrer's larger induction apparatus, and from 2—5 cells of Stoehrer's or Leclanché's galvanic battery. The increased irritability diminished with the subsidence of the chorea.

My experience disproves the applicability of these statements to all cases of chorea. In one case of hemichorea the faradic excitability of the muscles of the affected side was notably increased, but the reactions of the muscles and nerves to the galvanic current were equal on the two sides. In six other cases of hemichorea, which I examined with reference to these points, the electrical reactions were similar on both sides of the body. In only three of these patients, however, was I able to make the examination during the first period of the disease; in the others the chorea had lasted upward of a month.

Not infrequently heart-murmurs are heard, which—in the absence of previous rheumatism or other causes of valvular lesions, of hypertrophy or dilatation of the heart, and of other symptoms indicative of organic disease of this organ—should be regarded as merely dynamic in character. Some authorities consider such murmurs as purely anæmic, while others suppose that they are due to irregular choreiform contractions of the papillary muscles of the heart, which thus prevent the proper closure of the leaflets of the various valves at certain intervals. But this view is strongly contradicted by the fact that it is very doubtful whether choreic movements occur in the heart. As we shall see later, some authorities think that these choreiform movements of the papillary muscles are capable of giving rise to endocarditis, and explain in this manner the frequent occurrence of vegetations upon the mitral valves in fatal cases of chorea. At times, indeed, I have observed irregularity of the heart's action in patients suffering from chorea, but this has only been noticed in very anæmic patients, and therefore due to the condition of the blood, or it has occurred during violent choreiform movements of the respiratory muscles. In the latter cases it appears to me to be due to the irregular character of the respirations, the rhythm of which, as physiologists have taught us, exerts considerable influence upon the action of the heart.

The existence of chorea of the bladder is also a disputed point. The majority of neurologists deny that the involuntary muscular fibres are ever the seat of choreiform contractions. Van Buren and Keyes[1] mention three cases of chorea of the bladder which came under their notice, only one of which, however, is entirely conclusive, and which we copy in full.

Case II.—"Aged eight, is a fat, healthy, lymphatic boy; one of a large family of children, of whom nearly every male has distinct chorea, either generalized or affecting special muscles. Some of the older children have outgrown the tendency. The patient is troubled occasionally with slight general choreic twitchings, when from any cause his appetite is low, or his general health poor. Under such circumstances he has frequent paroxysms of intermitting, uncontrollable contraction of the bladder, forcing him to frequent micturition and attempts at emptying the bladder every few moments. Sometimes the call comes so suddenly that he wets his clothing, and he also is unfortunate at night. When the boy is enjoying good general health, neither his general chorea nor his fre-

[1] Genito-urinary Diseases with Syphilis, 1874, p. 231.

quent calls to urinate disturb him. He improves under arsenic, quinine, or any general tonic or country air."

I have also observed a case of chorea in which the patient, although previously able to retain his urine, was frequently seized with incontinence which came on suddenly, and which I could only explain on the hypothesis that the symptom was due to sudden, choreiform contractions of the detrusor urinæ. After the chorea subsided, the patient was again able to retain his urine as usual.

As a general rule, both pupils are considerably dilated in this disease, and do not respond readily to light. I have not observed any noteworthy difference in their condition in hemichorea. The appearance of the pupils is not, however, characteristic of this affection, and is frequently observed in children whose health is below par. Rosenthal reports one case in which, at the height of the paroxysm, he observed very marked dilatation of both pupils, which was not modified by exposure to a bright light or by introducing a small electrode between the sclerotic and conjunctiva. The pupillary dilatation disappeared spontaneously at the termination of the disease.

Dr. H. R. Swanzy [1] reports a case in which an ophthalmoscopic examination in a choreic girl (whose choreiform movements developed simultaneously with blindness of the left eye) showed the appearances indicative of embolism of the central artery of the retina. After the lapse of five weeks, the retinal circulation was restored, and the chorea likewise ceased about the same time. Three months later, this patient was suddenly seized with complete paralysis of the seventh nerve on the right side. Five days afterward, violent and persistent vomiting occurred, which continued all night and the following morning, and appeared to be of a cerebral character. The sense of taste on the right half of the tongue was somewhat impaired. Three months after the beginning of the paralysis the child was growing stronger and the paralysis had improved considerably. Dr. Swanzy regarded the paralytic affection as indicative of an organic cerebral lesion. It is unfortunate that no note was made of the electrical reactions of the affected nerve and muscles; but from the fact that the paralysis was said to be complete, and that the sense of taste was impaired, we should regard the former as more probably peripheral in its origin.

Some authors state that sensory disturbances are quite common in chorea, while others barely mention their occurrence. The French writers especially maintain that hemianæsthesia is a frequent concomitant of hemichorea, and that in some cases the special senses are affected. Although my attention has been directed to this point for several years, I have not been able to verify the observation. It is more than probable that many of the cases of chorea in which hemianæsthesia has been a prominent symptom, have been really examples of post-hemiplegic chorea, and therefore belong to another category. At other times, general hyperæsthesia of the surface has been noticed, or vague, wandering pains in the muscles and joints. Especial importance has been attached to tenderness upon pressure over the spinal column, particularly in the cervical and upper dorsal regions. Stiebel, Rosenbach, and Seifert have laid great stress upon the value of this symptom from a pathological and

[1] Ophthalm. Hosp. Rep., Sept., 1875.

therapeutic standpoint. Rosenbach showed (and I have verified his statement on several occasions) that the passage of a constant galvanic current through the spinal column will reveal the presence of tender points, at times when they are not appreciable upon pressure with the finger. But these tender points are not, by any means, invariably present in chorea, and some writers have been unable to detect them. If we bear in mind the great frequency of this symptom in hysteria, spinal irritation and general nervous prostration, we will not be tempted to lay much weight upon its appearance in chorea. I know, from personal observations, that physicians will sometimes regard this symptom as present when more careful examination shows that the position of the tenderness varies from time to time during the course of a single examination, is absent when attention is diverted, etc.

Mental disturbances are rarely absent in chorea. As we have mentioned in the beginning of this article, the children usually become peevish, irritable, and obstinate, at the onset of the affection. We desire to call attention emphatically to these symptoms, since they are almost invariably present, whether the disease be mild or severe, and because, in our judgment, they furnish important indications of the position of the lesion in the affection under consideration. But in rarer instances, true insanity develops during the course of chorea. Arndt,[1] who called attention to the close relationship which exists between the two diseases, states that many cases of insanity merely represent the transmission of the chorea from the motor to the intellectual centres of the brain. Insanity develops more frequently during chorea than is usually supposed. Krafft-Ebing[2] divides this form into three classes, viz., mania, melancholia, and a variety characterized by delusions of demonomaniac persecution. He regards them as "inanition-psychoses," caused by exhaustion induced by the violent character of the muscular movements, and by the diminished sleep. It is more than doubtful, however, whether these views will hold good with regard to all cases of this character, and one of my cases, at least, can not be included in this category. The insanity commonly occurs at the height of the choreic affection and the mental manifestations present a disjointed character, furnishing an analogue, as Arndt pointed out, to the phenomena observed in the muscles. Chorea sometimes simulates insanity although the mental powers are intact. Thus, the patients may appear to be incoherent in their speech, owing to the fact that articulation is interrupted by choreic movements of the muscles of phonation, giving rise to the involuntary utterance of words or disjointed sentences, foreign to the matter under discussion. This apparent incoherence of ideas, together with the destructive tendencies manifested by the patient on account of his lack of control over the muscles, has led physicians, in several instances, to make a diagnosis of insanity although the intellectual manifestations were entirely normal. An English alienist, whose name escapes me, has reported two cases of this nature which had been committed to an insane asylum. The ordinary course of choreic mania is exemplified by the history of the following patient under my charge.

Case III.—Fanny M., æt. 17½ years; family history is entirely negative as regards any neuropathic tendencies. The patient always enjoyed

---

[1] Arch. f. Psych. Bd. I.
[2] Handb. f. Psychiatrie. Bd. I., 1879.

good health until the beginning of the present ailment. Last August (1878), the patient caught cold while menstruating, causing a sudden arrest of the menstrual discharge. A week later, she began to have choreiform twitchings upon the right side of the body, and these soon increased in severity. After the lapse of a month, the left side also became involved, but the movements have never been so violent as upon the right side of the body. Toward the end of September, the patient had a bad attack of "nightmare," and during the next five or six weeks, gave marked indications of insanity. For some time she refused to eat, stating that her food was poisoned, and that she was being persecuted by strangers. She evinced hostile feelings toward her family, especially toward the mother, whom she called vile names, etc. At times she was unable to recognize her relatives and acquaintances, and thought that her female relations were men dressed in women's clothes. The patient was also vicious and violent, destroying her clothes and articles of furniture. During this time the choreic movements became extremely severe and continued even during sleep, so that the patient's limbs had to be tied down in bed. The muscular twitchings then rapidly improved and the symptoms of insanity disappeared at the same time. The chorea did not, however, stop entirely and was present to a moderate degree when the patient first came under my observation. I then prescribed Fowler's solution, beginning with five-drop doses three times daily, and increasing rapidly until twelve-drop doses were taken. Within a week after beginning this plan of treatment, the menses, which had been suppressed since last August, returned, and within a month the choreiform movements were no longer noticeable.

June 19, 1879, the patient returned, stating that the choreiform movements were returning on the right side of the body. She was again put on the use of Fowler's solution, gtt. viij., t. i. d., and rapidly improved. Apart from a certain amount of fretfulness and slight loss of memory, there were no mental disturbances during this attack; the muscular twitchings were also of a comparatively mild character.

In this case the mental aberration was most marked during the height of the affection, when the choreic movements were so violent that they did not even cease during sleep, and necessitated the application of stout bandages to the patient's trunk and limbs in order to prevent her doing injury to herself. As in the case just reported, the prognosis of this form of choreal mania is good. In the majority of instances the mental disturbances disappear as soon as the other symptoms have subsided, or within a couple of months afterward. Cases of maniacal chorea furnish, however, a large contingent of the mortality in this disease, not so much on account of the complication with insanity, but because the choreic movements are so severe that they interfere with sleep and nutrition. In other individuals, also, the manifestations of insanity persist, despite the disappearance of the choreiform movements and a return of the patient, in other respects, to a state of health. The following observation furnishes a good example of this nature:

Case IV.—Wm. K., æt. 8 years; patient's great-grandaunt was insane; grandfather died of apoplexy; a granduncle was an inebriate; a brother suffers from epilepsy; the mother is nervous and hysterical, but states that this condition has only developed in late years from worry and distress connected with the health of her children; no member of the family has ever suffered from rheumatism. The patient had measles and whooping-

cough during infancy; when three weeks old, he suffered from pneumonia and had a number of convulsions during his illness; also had two convulsions at the beginning of the attack of measles. At the age of 3 years and 5 years he had acute articular rheumatism. He also had another attack of rheumatism about 18 or 19 months ago, which lasted a week. Two years ago he began to have choreiform twitchings, which grew worse in the spring and almost entirely disappeared after the lapse of a year. But even at the present time considerable muscular twitching becomes apparent if the patient is very much excited; when he is calm the choreiform movements are not noticeable.

Last summer (1878) the patient began to act strangely. While at school he became extremely insubordinate, and displayed evidences of very bad temper. Upon one occasion he cut a playmate with a knife on account of a trifling dispute. During last September he began to entertain the delusion that objects around him were placed crooked. While sitting at table, he would carefully smooth out the wrinkles in the tablecloth, was continually moving the dishes in order to make them straight, and stated that the chairs and pictures were crooked. Finally, he ate from a low bench placed upon his knees, "on account of his inability to get the table straight." About the same time, he began to manifest an aversion to his mother, to whom he had been fondly attached. During the summer he was continually finding fault with his clothes—at one time the sleeves were too short, and then too long. Finally, he began to tear off his clothes and would run around naked, stating that the garments hurt him. The patient also had delusions of sight and hearing. Sleep was very much disturbed unless hydrate of chloral was administered. The appetite was very capricious, so that at times he would eat gluttonously and then again would lose all desire for food. The patient would wake up very tired in the morning; he has never had any epileptic fits during the day, nor have any been noticed at night (slept with his father who is a very light sleeper). He has gone to the window several times and threatened to jump out; also threatened to commit suicide by cutting himself with a knife. Has stated that he would rather be dead than alive, and remarked to his mother "that she would also prefer death if she felt like him, though he does not suffer from headache." Upon being questioned he is either unwilling or unable to state the character of his suffering. His insane condition is not constant, but alternates with lucid intervals which are more frequent and longer than they were last year.

May 15, 1880.—The patient has been under my observation since the summer of 1878, and, although the mental symptoms appeared to improve for a time, they have presented a relapse during the last six months, so that his condition is almost the same now that it was two years ago. The chorea, which had entirely disappeared, has begun to develop again since the beginning of this month.

Chorea is also associated at times with other convulsive affections, especially epilepsy. More frequently, however, we find that epilepsy develops in other members of the family. In rarer instances, a child who was choreic in early life, becomes epileptic in early manhood and finally presents evidences of insanity. Such a condition is almost invariably indicative of a severe hereditary neuropathic tendency. Among the cases under my observation, only four were complicated with epilepsy. The chorea may follow the epileptoid seizures, or may precede them by a variable period. Epileptiform seizures may also occur during the progress

of the chorea, as occurred in one of my patients, who presented the following history:

Case V.—Ellen D——, æt. 17 years, family history entirely negative as regards any hereditary tendencies. During childhood the patient had whooping-cough and measles, and passed through an attack of scarlatina at the age of 9. The menses appeared at the age of 13 and have been regular and normal up to the present time. The patient has suffered from left hemichorea for the past five or six years; the disease developed gradually and without any known cause. Upon repeated inquiry, it is found that the patient has masturbated almost daily since she was four years of age. The choreiform movements present a moderate intensity and are exclusively confined to the left side of the face and body. They have continued uninterruptedly since the beginning of the disease, disappearing during sleep. The muscular reactions to the faradic current are equal on both sides of the body. The measurements of the arms are alike and there is no loss of power upon the affected side; sensation is also normal. About four years ago, the patient began to have "weak spells" (*petit-mal*), during which she became dizzy, weak, and unconscious. She sometimes wakes up in the morning feeling tired and worn out (possibility of nocturnal epilepsy). She has on an average about one epileptic attack per month, but its development does not appear to be related in any manner to the period of menstruation. During the last six months the patient's memory has become somewhat impaired, and during the past two months she has become quite cross and irritable. At times, she experiences a sharp pain in the præcordial region, and is forced to stand still and hold her breath until the pain subsides. These attacks are not accompanied by a feeling of terror or by pain or numbness in the left arm. Upon physical examination, the apex of the heart is found a little to the left of the nipple and a slight thrill is felt. The valvular sounds are, however, perfectly sharp and distinct.

In this case, although the choreiform movements have continued for a number of years and were always confined to one side of the body, the absence of headache, eye-trouble, disorders of the cerebral nerves, and of paralysis of motion or sensation in the limbs, precludes the idea of an organic lesion as the cause of the chorea and epilepsy. It is much more probable that both affections are simply functional, and that the more severe neurosis (epilepsy) is due, in the absence of any hereditary taint, to the long continuance of the habit of masturbation, added to a primary (perhaps congenital) increased irritability of the nerve centres.

It is probable, also, from the history of the other cases of this nature which have come under my observation that they were due, in part at least, to excessive masturbation. Three cases occurred in unmarried females, and one in a young widow, all of whom confessed to the frequent performance of self-pollution. As we shall see later on, however, it is not an easy matter to determine the influence of the secret vice in the production of functional nervous diseases.

The chorea of pregnancy (chorea gravidarum) demands a few special remarks, on account of certain peculiarities connected with it. Perhaps the larger proportion of cases of chorea occurring in adult life belong to this category, though even in pregnant women the disease is of infrequent

occurrence. Barnes,[1] who made a very careful analysis of the literature of the subject, was only able to collect 56 cases. Bodo Wenzel[2] collected the histories of ten additional cases which had been reported in the journals from 1869—1874.

I have had no personal experience with regard to this complication of pregnancy, and shall, therefore, merely give an abstract of the conclusions arrived at by Barnes and Bodo Wenzel. I may state that, with the exception of the statistics compiled by these writers, very few cases have been reported.

The majority of cases occur in primiparæ between the ages of 20—25 years. Among 57 cases, 22 occurred in the first three months of pregnancy, and 23 from the fourth to sixth months. In very rare instances, the chorea makes its first appearance after delivery has been accomplished. In 14 cases out of 66, the patients had previously suffered from one or more attacks of chorea. The chorea of pregnancy is especially remarkable for its fatality. Thus, there were 18 fatal cases among the 66 collected by Barnes and Wenzel, or more than 27 per cent. In the fatal cases, the disease usually begins suddenly with great intensity and is often accompanied by considerable febrile disturbance, or by maniacal attacks. Death is generally due to exhaustion produced by the violence of the choreic movements and the loss of sleep and deprivation of nutrition attendant upon the maniacal excitement. The disease appears to have little or no effect upon the termination of pregnancy. As a rule, however, the choreiform movements rapidly disappear after the delivery of the child. In 7 cases the onset of the affection was attributed to fright; in 7 other cases, also, the disease was preceded by rheumatism and endocarditis.

The occurrence of chorea in one pregnancy appears to predispose to its recurrence in succeeding ones. We shall refer to the therapeutics of this variety of the disease under the general head of treatment.

Post-hemiplegic Chorea.

In concluding the clinical history of chorea we shall give a short description of the allied affections, known as post-hemiplegic and præ-hemiplegic chorea, and athetosis. Weir Mitchell[3] was the first to call especial attention to the form which he aptly termed post-hemiplegic chorea, though Trousseau,[4] in his article on chorea stated that "in some still rarer instances paralysis (I do not mean a mere diminution of muscular strength, but true paralysis), precedes the manifestation of convulsive phenomena." Although some advances have been made in the pathology of post-hemiplegic chorea, very little has been added to our knowledge of the clinical history of the affection since the publication of Mitchell's article. My own experience has been entirely confirmatory of the views advanced by this author.

Clinical history.—This affection is a hemichorea occurring at a longer or shorter interval, after the development of a cerebral hemiplegia, and always occupying the same side as the motor paralysis. The period of

---

[1] Obstetrical Transactions, vol. x., 1869.
[2] Schmidt's Jahrb., 1874.
[3] Amer. Journ. Med. Sciences, 1874.
[4] Lectures on Clinical Medicine.

its development varies (usually from a month to a year after the onset of the paralysis), and the choreiform movements generally make their appearance after the power of motion in the limbs has been considerably restored. It has also been found that the affection is almost invariably attended with a slight amount of contracture in the paralyzed limbs. The character of the movements does not differ from that of ordinary chorea, except that they are more apt to be absent while the patient keeps the limb quiet, and, like choreic twitchings, they cease during sleep. It is a curious fact that, while the affection is comparatively rare after the hemiplegia of adults, it is very common in the cerebral hemiplegia of infancy and childhood, as had been noticed by Weir Mitchell. I may remark here as an interesting circumstance, that cerebral hemiplegia of infancy and early childhood, when not followed by post-hemiplegic chorea, almost always becomes complicated with epilepsy or hystero-epileptiform seizures. To this rule I have found very few exceptions.

In the large majority of cases post-hemiplegic chorea follows cerebral hemorrhage, although it may also occur in the course of hemiplegia due to embolism, thrombosis, tumors, cerebral atrophy of childhood, or abscess of the brain.

According to French authors, especially Charcot[1] and Raymond,[2] the motor disturbances of post-hemiplegic chorea are frequently combined with sensory disorders. Thus, among 30 cases of this affection collected by Raymond (chiefly from the wards of La Salpêtrière), 10 were complicated with hemianæsthesia. The anæsthesia not alone affects the general sensibility, but may also involve all of the special senses. Among my own cases I have been unable to discover any disorders of sensation. I should, however, state that I have chiefly observed this affection in young children, in whom it is very difficult to obtain any accurate data with regard to sensation.

Among 10 cases analyzed by Gowers[3] hemianæsthesia was only observed four times. Hemiopia was present in two of these cases, but in one of them it was on the side opposite to the hemiplegia, and was evidently due to a distinct lesion.

Pre-hemiplegic chorea is similar in its clinical characters to the post-hemiplegic variety, varying only with regard to the date of its appearance. It usually occurs from a few hours to several days before the full development of paralysis, and subsides as soon as the hemiplegia becomes well marked or complete.

After the paralysis has improved, the choreiform movements may again return and run the same course as the post-hemiplegic variety. Pre-hemiplegic chorea is much more infrequent than the kindred affection.

Charcot and his followers insist very strongly that the motor affection under consideration is characteristic of a lesion situated in a well-defined portion of the brain, viz.: the posterior part of the internal capsule and of the optic thalamus and caudate nucleus. Although Raymond has shown that this is very generally the case, investigations by other authors have disproven the universal application of this statement.

Thus, Weir Mitchell found a lesion of the corpus striatum in two cases of post-hemiplegic chorea, and Gowers observed a cicatricial induration

---

[1] Leçons sur les maladies du système nerveux, 1877.
[2] Étude anatomique sur l'hémichorée, etc., 1876.
[3] Med.-chir. Trans., 1876.

of the optic thalamus, extending across its centre beneath the upper surface and approaching at its outer part, but not involving, the ascending white fibres from the crus cerebri. In a case reported by Magnan [1] a hemorrhagic extravasation as large as a small hazel-nut was found in the left cerebral peduncle, at its insertion into the optic thalamus.

The prognosis in these affections is very poor indeed. Gowers reports a case of complete recovery after the employment of the constant galvanic current for a number of months. I have been less fortunate in my experience with this agent, as a slight amount of improvement has been the utmost which I have been able to obtain.

Charcot regards the prognosis as absolutely unfavorable, and states that the choreiform movements only terminate with the patient's life.

Athetosis.

Closely allied, and by many considered as identical, with these affections is the disease which was first described by Hammond under the title of athetosis.

The main features of this affection consist of slow and involuntary muscular contractions, occurring especially in the hands and feet, and producing rhythmical alternations of flexion and extension in the fingers and toes. The disease is usually unilateral in character (the right side has been involved in most cases), but in some instances, both sides of the body have been affected. The majority of cases have been associated with other cerebral diseases, such as epilepsy, dementia paralytica, locomotor ataxia, idiocy, etc.

The following is the history of an interesting case of this affection, which is probably unique from the fact that the athetotic symptoms are the sole evidences of disease, there being absolutely no other manifestations of a cerebral affection.

Case VI.—G. W. L——, æt. 18 years, no occupation. The patient's mother died of phthisis, the father and other members of the family are living and healthy. According to the father's statement, the delivery of the patient was effected normally, though it occupied a somewhat longer period than that of the other children; no resort was had to the use of the forceps. The father noticed a peculiarity about the fingers of the right hand (he was unable to open them as readily as upon the opposite side) within two or three days after birth.

Present condition.—The patient is a very well nourished and bright lad. His education has been neglected on account of his infirmity, but his mental powers are active and fully equal to the average of boys in his station of life. The special senses are perfectly normal. The right side of the face appears, perhaps, to be slightly atrophied, and the mouth droops a trifle on this side. When the patient is watched and when he talks or smiles, the facial muscles upon the right side, especially those inserted into the angle of the mouth, are in an almost continual state of clonic and rather slow contraction.

Upon measurement, it is found that the length of the arms from the acromion process to the end of the radius is equal on both sides. The right arm measures 8⅜ inches in circumference, the left arm, 9⅛ inches;

[1] Gaz. méd. de Paris, 1870.

the right forearm measures 8⅞ inches; the left, 9½ inches. In the usual position of the limb, the forearm is somewhat flexed, and the hand and fingers are also forcibly flexed. During rest the muscles of the arm are quite flaccid, but as soon as the patient attempts to move the arm in any direction, the muscles become as rigid as wood. The patient usually grasps the affected hand with the other in order to keep it quiet.

If he sets the hand free, the deltoid of the affected arm immediately contracts, drawing the limb away from the chest, and the fingers begin to twitch slowly, the most common form of movement being toward more marked flexion, alternating sometimes with slow extension, especially of the 2d and 3d phalanges.

If the patient is directed to pick up anything with the right hand, he must first extend the fingers somewhat with the left and then, as he makes the attempt, the fingers become hyperextended and widely divergent, so that it is impossible for him to grasp an object.

The arm can be moved quite freely at the shoulder-joint, though not as forcibly as on the sound side; passive motion at this joint meets with slight resistance, which is unattended with pain. After a great deal of effort, the patient becomes able to flex the forearm on the arm after it has been extended; when extended the triceps is exceedingly firm and contracted, the biceps not to the same degree. While the patient is flexing the forearm the biceps becomes flabby, and when it is completely flexed, this muscle is entirely flaccid. The forearm cannot be voluntarily extended after it is flexed, nor can the hand be flexed after it has been extended. It requires a considerable exertion of muscular power on my part in order to vary the position of the hand from any in which it may be at the time of the experiment. The continuous movements of the fingers, when not supported by the other hand, prevent the patient from employing it for any purpose whatever, and render even dressing inconvenient and troublesome.

The lower limb is not affected to the same extent as the upper. The right thigh measures 16¼ inches, the left 18 inches; the right calf measures 12 inches, the left 12½ inches. The first phalanges of the foot are flexed, the second and third are hyperextended. When the patient attempts to walk, the gastrocnemii and solei become contracted and very rigid, pulling up the heel so that he is forced to walk on the front part of the sole of the foot; at the same time, the tibialis anticus contracts, drawing up the inside of the foot. When the patient is directed to move his toes, slow movements of extension and flexion, similar to those observed in the fingers, though of course not so marked, become evident. Walking is not very much interfered with, as the involuntary movements of the toes are partially restrained by the shoe.

Cutaneous sensibility and the electrical reactions of the muscles are normal throughout the entire body. The movements of the muscles continue unabated during sleep. The patient is perfectly healthy in all other respects. The treatment consisted in the application of the faradic current to the muscles of the arm (this was merely done to retain the patient under observation), my galvanic battery not being in working order. To my great surprise, the abnormal facial movements disappeared almost entirely in the course of 5 to 6 weeks (the current had never been applied to the face), and the violence of the movements of the fingers was also slightly diminished. I then applied the constant galvanic current for a couple of months, but without producing any further improvement. The patient then passed out of my observation.

Very few opportunities have been afforded for investigating the pathological anatomy of this affection, and I have only been able to obtain records of five post-mortems upon patients suffering from athetosis. In one, the brain was found normal; in the second, two small spots of softening were discovered in the first temporal convolution; in the third case, the patient also suffered from dementia paralytica, and the brain presented no evidences of a localized lesion; in the fourth case, the athetosis occurred as a complication of locomotor ataxia, and a small spot of softening was found at the posterior, inferior, and outer extremity of the right lenticular nucleus. There was probably, however, no connection between this lesion and the athetosis, since the latter occurred upon both sides of the body. The fifth case is of great interest with regard to the light thrown upon the relations of the affection to post-hemiplegic chorea, and I shall, therefore, present a short abstract of the case as reported by Dr. Sturges.*

Case VII.—H. B——, æt. 33 years; when three years old, the patient had whooping-cough, and shortly afterward had two fits, which left him paralyzed on the left side. He gradually gained power, however, in the limb, and, at the age of ten could run about as well as other boys. The athetosis appeared soon after the fits, and gradually increased in severity as the muscular power was restored.

The movements were almost exclusively confined to the left upper limb, and were continuous and involuntary. When the hand was extended with the palm downward, the index and middle fingers were slowly and gradually flexed. The thumb was also adducted, the hand was then supinated, the fingers again extended, and the thumb abducted; pronation of the hand completed the cycle. The patient could slightly control the movements by a great effort of the will; the hand was only quiet during sleep. The left leg occasionally exhibited a somewhat similar condition, but only when the patient was tired out after a long walk. Death occurred from diarrhœa and exhaustion.

Autopsy.—Brain: right hemisphere distinctly smaller than the left; the posterior half of the middle and inferior frontal convolutions, and, to a slighter extent, the superior and ascending frontal were distinctly smaller on the right side than on the left; the parietal convolutions were also smaller on the right side. There was a depression on the anterior portion of the temporo-sphenoidal lobe, about one inch long; there was also a deep depression extending backward into the lobe, about three-fourths of an inch deep. A deep excavation was found between the anterior extremity of the perforated spot and the convolutions of the island of Reil, extending backward to the level of the corpora albicantia and forward to the anterior surface of the hemisphere. The sides of the fissure seemed to have been in apposition, except outside the perforated spot, where the cavity was about one-fourth inch wide; its roof was formed by radiating fibres spreading upward from the pons. Upon opening the ventricles, almost the whole of that portion of the right corpus striatum lying in front of the thalamus appeared to be destroyed; the posterior portion of the nucleus caudatus was unaffected. A small portion of the inner part of the corpus striatum near the middle appeared intact, but the whole of the gray substance was destroyed. The optic thalamus seemed to be quite healthy.

---

* Lancet, March 15, 1879.

These post-mortem investigations, especially the one last mentioned, appear to me to disprove conclusively Hammond's assumption that athetosis is a distinct disease.[1] The latter case demonstrates that, in some instances at least, the disease merely constitutes a peculiar variety of post-hemiplegic chorea (this should more properly be called symptomatic chorea). I have also distinct recollection of a case of post-hemiplegic chorea in a girl, æt. 9 years, in which the movements of the fingers were exactly similar to those described by Hammond as characteristic of athetosis. In this case, the patient, while suffering from intermittent fever three years ago, was suddenly seized with a convulsion which took the place of a chill. The child was found to be hemiplegic on the right side on the following morning. As the power began to return to the affected arm, the athetoid movements made their appearance and have continued ever since.

[1] Leube (Deutsch. Arch. f. klin. Med., Bd. XXV., p. 242, 1880) reports a case which also appears to show that athetosis may, in some instances, be a mere variety of ordinary chorea. The patient in question, after prolonged exposure to wet, developed athetoid movements throughout the entire body, which persisted steadily for four years. At the end of this time the symptoms changed to those of ordinary severe chorea, and continued as such during the entire period in which the patient remained under Leube's observation.

CHAPTER II.

ETIOLOGY.

ALMOST all writers are unanimous in the opinion that sex constitutes one of the chief predisposing causes of chorea, the female sex presenting by far the larger proportion of cases of this affection. In the following statistics, which include a very large number of cases, the proportion of males to females is 382 to 1,053, or nearly 1 to 3:

	Females.	Males.	Total.
Sée	393	138	531
Rufz	138	51	189
Hughes	240	69	309
Steiner	40	12	52
Pye-Smith	106	42	148
Peacock	86	37	123
Author	50	33	83
	1,053	382	1,435

The majority of cases of chorea occur from the fifth to fifteenth years of life, as is shown by a glance at the following tables:

	Below 5 yrs.	5—10 yrs.	10—15 yrs.	15—20 yrs.	Above 20 yrs.
Sée	11	94	57	17	12
Steiner	4	46	6	..	..
Pye-Smith	5	62	44	19	6
Peacock	..	22	51	14	5
Author	2	27	28	9	5
	22	251	186	59	28

In very rare instances, chorea occurs from birth. After careful examination of the literature of the question, I have only been able to find the histories of 8 cases of congenital chorea, reported by Heller,[1] Mayo,[2] Monod, Spamer,[3] Althaus,[4] Fox,[5] and Richter (2).[6] In Mayo's case the mother had been extremely nervous for a period of two months after a disgusting object had been thrown upon her bosom (while she was four months advanced in pregnancy). In Richter's two cases, the mothers had been very much frightened shortly before confinement. Spamer men-

[1] Wien. med. Wschr. 19. 1876.
[2] Outlines of Human Pathology, p. 170.
[3] Wien. med. Wschr. 52. 1876.
[4] Diseases of the Nervous System, 1878.
[5] Brit. Med. Journ., 1873, No. 653.
[6] Sitzb. d. Dresden Ges. f. Nat. u. Heilk. Jan. 5, 1867.

tions, with regard to his case, that the mother had been very nervous and depressed during the entire period of pregnancy, on account of the death of one of her children. No reference is made in the reports of the remaining four with regard to the probable exciting cause of the disease.

Chorea is very infrequent during adult life, and becomes extremely rare in old age. When it occurs after the age of fifty, it is usually attended with some mental derangement, especially dementia. Five cases have come under my observation which developed after the age of thirty-five years, one of which was complicated with insanity. In the *Lancet* for 1878, Dr. R. T. Wright reports the history of a case of chorea of three years' standing in a man seventy-eight years of age.

Heredity exercises but little influence upon the development of chorea, and among upward of ninety cases which have come under my observation, I have not been able to obtain a single instance of the transmissibility of the predisposition to the affection from parent to child. Not infrequently, however, it is found that one of the parents, usually the mother, suffers from nervousness or hysteria, and, in a considerable number of instances, I have observed that one or more of the brothers or sisters of a choreic patient were subject to epilepsy or hysteria.

Although I have not collected any comparative statistics on this question, I am nevertheless convinced that chorea develops in those families in whom the neuropathic tendency has not taken strong root, while in those which are more severely affected, more serious nervous diseases, such as obstinate neuralgias, epilepsy, insanity, etc., make their appearance. In not a few cases, however, chorea is combined with epilepsy, especially when the former affection has become chronic.

Anæmia also exerts considerable influence as a predisposing cause of chorea, but its importance as a pathogenic factor must not be overestimated. It is true that the majority of patients who have suffered from chorea for any length of time present symptoms of anæmia (pallor of face, conjunctivæ, and lips, irritable heart, shortness of breath, rapidly developing sense of fatigue), but this condition is then secondary to the disturbed sleep, the insufficient supply of nutriment, and the continual muscular effort to which the patients are subjected. Nevertheless, we not infrequently meet with patients of a very nervous temperament who manifest slight choreiform symptoms as soon as the general health, for any reason, sinks below the normal standard.

Onanism is also looked upon as an active predisposing cause of chorea, as it is of so many other nervous affections. I have, however, only been able to obtain evidence in a few cases of the sufficiency of this cause as an agent in the production of the disease. In fact, I am of the opinion that the baneful effects of the "secret vice" have been greatly overestimated by the profession at large as well as by the laity. The habit is undoubtedly practised to an enormous extent among children of both sexes, and if its potency as a disease-producing factor were as great as it is claimed to be by so many physicians, chorea, as well as other functional neuroses, would be much more common than they really are. While I therefore believe that excessive onanism, by lowering the healthful tone of the nervous system, may prepare the way for the more ready development of nervous affections, I doubt whether it is often the principal agent in their production.

The exciting causes of chorea are very numerous. Peacock[1] gives the

[1] St. Thomas' Hosp. Rep., 1877.

following analysis of ninety-two cases, coming under his own observation, with regard to causation:

Not ascertainable	23
Fright	25
Excitement	8
Blows on head	5
Driving a van in very hot weather	1
Prolonged lactation	1 or 2
Pregnancy	3
Suppression of the catamenia	2 or 3
Scarlatina	2
Rheumatism	8 or 9
Worms	3

Among the ascertainable causes, therefore, fright holds the chief place. As a rule, the development of the chorea follows this exciting cause after the lapse of from six to forty-eight hours. In four cases under my observation, the patients, who had been frightened during the night by an alarm of fire or by burglars, awoke the following morning with well-developed choreiform twitchings of the entire body. In one instance, the chorea developed immediately after the exciting cause. The patient, an anæmic and extremely nervous girl of ten, was suddenly startled last August by a terrible clap of thunder, which was so severe that it was the subject of town talk for several days. The guardian of the patient, a very intelligent young lady, assured me that the choreic movements developed immediately after the thunder-clap, and that they were very violent from the beginning. When I saw the girl on the following day, the movements were so violent that the little patient was unable to maintain her seat, and could only walk with difficulty. Violent fits of anger, or other causes of excitement, will sometimes act in the same manner as a severe fright. When the chorea develops one or two weeks after profound mental excitement, it is extremely doubtful whether this can, with propriety, be looked upon as the exciting cause. We must remember that the friends of the patient are usually anxious to aid us in our search for the origin of the disease, and that their zeal often leads them to mention, as causes, occurrences which long antedated the development of the muscular twitchings. It is, therefore, always well, when told that the chorea came on after a fright, fall, etc., to enter somewhat into the details of the case, and judge for ourselves whether there is really any connection between the two occurrences. Inattention to this simple rule has led me astray in not a few cases of this as well as of other diseases.

Among the other exciting causes of a mental nature we must mention imitation. This, however, is exceedingly rare, and has only occurred once in the cases which have come under my observation (I have previously referred to this case on page 3). Bricheteau reports a very interesting example of an "epidemic" of chorea which developed in his wards at the Hôpital Necker. Within a week after the admission of a choreic patient into the hospital, eight other patients (females) occupying the same ward became affected with the disease. Its further spread was only prevented by isolating the choreic patients.

Rheumatism constitutes one of the most interesting causes of chorea, both on account of the differences of opinion with regard to its importance and also on account of its relations to the pathology of the affection.

The opinions of different authorities vary diametrically with regard to this question. The theory of its intimate relations to chorea has been especially promulgated by English and French writers. M. Sée [1] states that one-half of the entire number of cases of chorea are caused by the rheumatic poison. Roger [2] is even more pronounced in his views concerning the relations of rheumatism and chorea than the preceding author, and thinks that the coincidence of these two affections is as much evidence of a pathological law as the so frequent coexistence of endocarditis and rheumatism. Among 104 cases which were reported in Hughes' and Brown's paper on chorea in the *Guy's Hospital Reports*, for 1855, and which were carefully investigated with regard to the previous existence of rheumatism, only fifteen cases were found in which the latter affection had not been present, or in which a cardiac murmur was not audible. Trousseau also believes that rheumatism is one of the most common causes of chorea. But numerous statements to the contrary have been made by various authorities. Steiner [3] found that among 252 cases under his observation, a history of previous rheumatism could be obtained in only four. Octavius Sturges [4] saw only five positive cases of acute rheumatism, among seventy-one cases of chorea. Among forty personal cases which I examined with great care in this respect, only three gave a history of previous rheumatism. Among these three patients, two presented a well-marked mitral systolic murmur, and in the third, the heart-sounds were normal.

It must be remembered, in estimating the importance of rheumatism as an etiological factor in chorea, that a diagnosis of the former disease is frequently made on insufficient data. The cases of acute rheumatism in children which have come under my observation, have presented exactly similar symptoms to those occurring in acute rheumatism in the adult. Not infrequently, however, neuralgic pains in the joints, muscular rheumatism in the neighborhood of the joints or hyperæsthesia of the skin, are regarded, in children, as sufficient evidence of rheumatism. In like manner many physicians look upon a basic heart-murmur, even when unattended with hypertrophy of the organ or with the subjective symptoms of valvular lesion of the heart, as sufficient evidence of endocarditis, and presumably, therefore, of antecedent rheumatism. In such cases, the history of previous pains in the limbs is regarded as ample testimony of the rheumatic origin of the endocarditis.

Rilliet has also referred to the fact that chorea rarely appears in certain localities in which acute articular rheumatism is of frequent occurrence, as, for instance, in Geneva.

As a rule, chorea, when connected with rheumatism, develops in the latter stages of this affection or at a varying interval after recovery. In rare instances, however, the chorea precedes the attack of rheumatism in point of time. Roger [5] mentions an extremely interesting case occurring in a girl æt. 11½ years who suffered, within less than five years, from six attacks of acute articular rheumatism and five of chorea, hemiplegia and endocarditis also developing during two of the relapses.

Chorea is also said to be sometimes caused by reflex irritation, such as the presence of worms in the intestinal canal, toothache from carious teeth, amenorrhœa, pregnancy, irritation of cicatrices in various parts of the body. There is no doubt that these causes may sometimes give rise

[1] Mém. de l'Acad. Nat. de Méd., t. xv.
[2] Gaz. méd. de Paris, Mar. 7, 1868.
[3] Prag. Vjschr. 1868.
[4] Lancet, p. 283, 1878.
[5] Arch. génér., vol. ii., p. 658.

to the disease, although the existence of reflex chorea has been denied by some authorities. Eulenburg states that he has, on several occasions, found the disease, when due to dental disorders, disappear after the extraction of the carious teeth, and again make its appearance upon the development of fresh trouble in the teeth. The case of Fanny M., which we described upon page 8, seems to show that chorea is sometimes dependent on the sudden abolition of the menstrual discharge, as the chorea appeared soon after the development of the amenorrhœic condition, and no other cause could be ascertained.

It is doubtful whether pregnancy produces chorea from the reflex irritation due to the presence of the fœtus in utero, or from the anæmia and hydræmia which usually exist in this condition. Judging, however, from the action of pregnancy in the development of other neuroses, I should think it probable that chorea gravidarum is attributable to reflex irritation.

Finally, we must consider the effects of syphilis upon the development of chorea. Although this affection is such a frequent cause of various nervous diseases, there are extremely few examples on record of cases of chorea which may be attributed to its agency. I have only been able to obtain records of seven cases,[1] one of which, however, was really post-hemiplegic chorea, a consideration of which has been previously entered upon.

Of these seven cases, four occurred in females and three in males. Two developed at the age of seven years, one at the age of twelve, and the remainder between the ages of twenty and thirty-three years. Five recovered completely under the use of anti-syphilitic remedies, one was unimproved, and one proved fatal.

It is extremely difficult to determine in what manner the syphilitic virus produces the choreiform movements. In all probability they may be attributed to disturbances in the nutrition either of the cortical motor centres or of the basal ganglia caused by the changes in the walls of the vessels which are now recognized as the frequent origin of syphilitic cerebral disturbances.

In very rare instances, chorea appears to be due to malarial influences, as in the following personal case.

Case VIII.—Mrs. Eliza G., æt. 26 years, married six months; her paternal uncle and father's first cousin were insane; the patient was in fair health until marriage (six months ago), but since then very marked hysterical manifestations have developed. Upon vaginal examination the hymen is found to be intact, and the patient, on inquiry, states that sexual intercourse has never been satisfactorily consummated. The hysterical symptoms were undoubtedly due to this condition of the genital organs, and to the nervous irritability produced by the ungratified sexual desire. For the past three months the patient has been suffering from muscular twitchings which were chiefly confined to the right side (face, arm, and leg), but were also manifested to a slighter degree on the left side. The patient and her mother are positive in their statement that the choreiform movements only appeared every other day during the first two months. Within the last month, the twitching was noticeable every day, but was very slight on the alternate days, so that distinct periodicity is marked even at the

[1] Zambaco: Des affections nerveuses syphilitiques, Paris, 1862. Phila. Med. Times, April 14, 1877. Alison: Amer. Journ. of Med. Sciences, vol. ii., 1877. Raymond: Étude, etc., sur l'hémichorée, etc., Paris, 1876.

present time. Upon examination, the spleen was found to be enlarged, but there were no other evidences of malaria. Acting on the theory that the chorea was of a malarial nature, I placed the patient on thirty-grain doses of quinine daily (November 19th), which was sufficient to produce marked cinchonism. On November 27th the choreiform movements had entirely disappeared, and did not return while the patient remained under observation (six to eight weeks). There was also marked improvement as regards the severity of the hysterical symptoms, but I attributed this change to the fact that the patient, at my advice, lived separately from her husband, thus removing, in part at least, the cause of the disorder.

CHAPTER III.

PATHOLOGICAL ANATOMY.

ALTHOUGH a great deal of attention has been devoted to this branch of our topic within the last twenty years, especially by English pathologists, there is still considerable room for further investigation. The autopsies which were made in the early part of this century are almost entirely valueless, as they were performed before the modern improved methods of pathological and histological research had come into general vogue. But this criticism will also hold, to a certain extent, with regard to the autopsical investigations made at the present day. Numerous cases are reported in the journals in which the writer has felt himself justified in declaring the nervous centres intact, although microscopical examination has been entirely omitted.

The opportunities for post-mortem examinations in this affection are comparatively rare on account of the infrequency of a fatal termination (among upward of ninety cases which have come under my observation, in only one did a fatal result follow), and it is therefore to be hoped that those observers who are enabled to make such investigations, will resort to a careful microscopical and macroscopical examination of the central nervous system, as well as of the peripheral nerves.

Aitken examined the specific gravity of the basal ganglia of the brain in a case of chorea, and found that that of the corpus striatum and optic thalamus on the right side was 1·025, and on the left side 1·031. These figures vary considerably from those furnished by Bucknill for these ganglia in healthy brains (1·036).

Kirkes[1] reported several cases of fatal chorea in which he found endocarditis present upon autopsy. Small, fine granulations were present upon the valves of the heart, especially upon the mitrals. Numerous observations of a similar character have been made by other observers, and even in cases in which no symptoms of rheumatism had been presented by the patient during life.

Broadbent[2] mentions the following case. The patient was a young woman, twenty-three years of age, who began to manifest a change in disposition two years previously. Her general health soon became impaired, and of late it was noticed that the patient's skin was assuming a browner hue. She was confined to her bed for a period of two weeks with what was called a "low fever," and upon her recovery from this condition it was found that she had lost the perfect control over the movements of the left arm. Well-marked choreiform twitchings soon appeared in this member, then spread to the left leg, and finally to the limbs on the opposite side of the body. When admitted to the hospital, the patient was very feeble and presented general choreiform movements, which were

[1] Med. Gazette, 1850, and Med. Times and Gazette, 1863.
[2] Trans. Lond. Path. Soc., vol. xiii., 1861.

most marked upon the left side. The chorea continued until death, which occurred ten days after admission.

Post-mortem.—We will merely mention the appearances presented in the nervous system, the other lesions having no relation to the chorea. The brain was perfectly healthy in appearance and consistence. The spinal cord was firm and healthy except at the posterior surface of the dorsal region, two inches above the lumbar enlargement. In this situation a small tumor was noticed, which was at first supposed to spring from the surface of the cord, but, upon section, was found to originate from the interior.

Tuckwell[1] gives the following results of the post-mortem examination in a boy æt. 17 years, who suffered from acute chorea, which had become complicated with mania and proved rapidly fatal. *Autopsy.*—Upon the middle of the upper surface of the right cerebral hemisphere was a spot of softening which appeared to be limited strictly to the cortical layers. A similar spot was found upon the under and outer aspect of the posterior lobe of the same hemisphere, but in this situation the lesion also extended a certain distance into the white matter. Upon carefully tracing up the posterior cerebral artery, an embolus with a secondary thrombus was found in one of the branches which led in the direction of the softened spot. A spot of softening of considerable size was also found in the dorsal region of the spinal cord, together with a small embolus in one of the vessels within the affected district. The heart appeared healthy with the exception of the auricular aspect of the mitral valves, which presented a row of small, delicate vegetations of recent origin.

In the "Saint Bartholomew's Hospital Reports," vol. v., Tuckwell mentions the following interesting post-mortem results in a fatal case of chorea: The brain was well developed and presented nothing abnormal in the meninges or upon the surface. Upon tracing out the posterior cerebral artery, and thereby displacing the middle cerebral lobe, the inferior portion of this lobe on the right side was found softened. The softening involved the deeper layers of the cortex, and more especially the subjacent white substance; it also extended upward into the neighborhood of the corpus striatum and optic thalamus. The basal ganglia, however, appeared to be normal. The corresponding portions of the left hemisphere were also softened, but to a less extent than on the right side. The remaining portions of the brain presented a remarkably firm and healthy appearance.

The spinal cord appeared to be normal in all respects.

Heart.—The auricular aspect of the mitral valves showed the following changes: a line of numerous bright, clustering, warty vegetations, some as large as a pin's head, others barely visible, extended in a sinuous course all along the free margin of each leaflet. In two or three places, the little growths hung only by a fine pedicle to their line of attachment.

Wilson Fox[2] also reports a case of chorea with cerebral embolism. During the course of the disease the patient had presented a mitral murmur, but no history of previous rheumatism could be obtained. Upon autopsy, the heart was healthy, except that the free borders of both flaps of the mitral valves were thickly covered with rough granulations, which were apparently of recent origin. The brain appeared perfectly healthy to the naked eye, but Bastian, who made a microscopical examination of

[1] Brit. and For. Med.-Chir. Rev., 1867.
[2] Trans. Lond. Path. Soc., xx., 1869.

this organ, observed plugging of some of the blood-vessels in the medulla oblongata.

Magnan[1] reports the following case: C. J., æt. 73 years; previous history unknown, presents choreiform movements of the right arm and leg; talks incoherently. After great difficulty, it is found that this condition has only lasted three or four days. No accurate data can be obtained with regard to sensation.

Upon the day after admission to hospital, the movements were incessant; they extended to the neck and face, but only involved the right side. They increased during excitement, and ceased during sleep. Upon the following day the choreic movements had increased in severity, the face was grimacing (to the right) and the arm and leg were in constant motion. At 1 P. M. the patient became pale and then cyanosed, the movements ceased, and death occurred suddenly in syncope.

Autopsy.—The cerebral meninges were thickened, opaline in places, and were readily detached from the convolutions. The convolutions were yellowish and diminished in size. The vessels at the base were atheromatous; the ependyma of the lateral ventricles was thickened. The état criblé was visible in both hemispheres, especially in the corpora striata and optic thalami.

An incision through the left cerebral peduncle, at its insertion into the optic thalamus, showed a hemorrhagic foyer as large as a small hazel-nut, composed of soft, reddish clots of recent date; the adjacent nerve-tissue was torn and infiltrated with fluid. The pons and medulla presented no appreciable change. A yellowish patch of old softening was found upon the inferior surface of the left lobe of the cerebellum.

W. Howship Dickinson[2] gives the results of autopsical examination in seven fatal cases of chorea, the appearances in which may be briefly summed up as follows:

Case I.—Injection of all the vessels of the brain and cord, especially in the corpora striata and dorsal region of the cord. Hemorrhage into the central canal of the spinal cord, which was distended by serum.

Case II.—Similar injection of the brain, superficial hemorrhages and exudation around the arteries of the corpus striatum. Injection of the spinal cord.

Case III.—Injection of cerebral vessels, especially around the optic thalamus. Injection of the cord and hemorrhages into the gray matter of the dorsal and lumbar regions.

Case IV.—Injection of the brain, chiefly venous, and of the corpora striata. Injection of the cord, with large hemorrhage into the cervical gray matter.

Case V.—Venous injection of the brain, especially of the corpora striata; arteries in the convolutions near the Sylvian fissure surrounded by blood crystals. Injection of spinal cord.

Case VI.—Recent injection of the structures at the floor of the lateral ventricles, and of the spinal cord.

Case VII.—Spots of "sclerosis" (simple atrophic degeneration) in the substantia perforata and in the gray matter of the corpora striata. Extensive exudations into the cord.

The heart was examined in six of these seven cases. Vegetations

---

[1] Gaz. méd., Paris, 1870.

[2] Med.-chir. Trans., vol. lix.

upon the mitral valves were only found absent in one case (a widow, æt. 54 years).

Elischer [1] found anatomical changes in all parts of the nervous system in a patient suffering from chorea gravidarum, who died of puerperal endometritis; the median and sciatic nerves (which were the only peripheral nerves examined) were flattened and smaller than normal, and had, at the same time, assumed a firmer consistence. Microscopical examination showed that the amount of interfibrillary connective tissue was greatly increased, and that it contained a large number of spindle-shaped nuclei. Numerous small extravasations of blood were visible between the nerve-bundles. In some of the nerve-fibres the sheath of Schwann appeared cloudy, in others it presented a vitreous appearance; in these fibres, the axis-cylinders could only be detected with difficulty.

Spinal cord.—The walls of the blood-vessels, especially the tunica adventitia, were thickened and contained numerous nuclei. The central canal contained considerable serum, and the surrounding connective tissue was firmer and more abundant than usual. The four principal collections of ganglion cells in the gray matter were separated from one another by well-marked bundles of newly formed fibrous tissue, which, in the substantia gelatinosa, contained large numbers of nuclei. The ganglion cells were misshapen and did not stain well with carmine. The nuclei of these cells were no longer visible.

Brain.—The vessels of the basal ganglia presented the appearance of fatty, amyloid, and pigment degeneration. The vessels were covered, in certain places, with granular cells. Especially in the corpus striatum. Elischer attaches no importance, however, to this appearance, as he observed similar lesions in two patients who had died of tuberculosis and internal hemorrhage. The small vessels, especially in the convolutions, contained numerous emboli, composed of red and white globules. The neuroglia of the brain was increased in amount and firmness, and was strewn with large, coarsely granular nuclei. The vessels of the corpus striatum were surrounded by numerous minute deposits of a rusty-brown pigment. The ganglion cells contained such a large amount of pigment that the protoplasm appeared to be entirely destroyed and displaced; some cells contained no nuclei, and were filled with fat granules. The changes in the ganglion cells were only visible in the basal ganglia, the cerebellum and cerebral convolutions being normal.

The following autopsy, for an account of which I am indebted to the kindness of Dr. E. A. Maxwell, Curator to Charity Hospital, was held upon a patient whom I had observed from time to time, for a period covering several years. The patient was an inmate of the Epileptic and Paralytic Hospital on Blackwell's Island.

Case IX.—Catherine Eppersault, æt. 78 years, admitted 1869, died November 8, 1878. Patient has been choreic for more than twenty years. Nothing is known about her previous history as she had no friends and her speech was so much impaired that it was impossible to get a history from her. Such language that she used, and which could be understood, was vile and profane. The choreic movements affected the face, tongue, trunk, and extremities, and were extremely violent in character. The body and limbs underwent the most grotesque and hideous contortions, the facial

[1] Virch. Arch. Bd. 61, p. 485, and Bd. 63, p. 104.

muscles were in continual activity, and the tongue was protruded and retracted with considerable force. The movements were very much increased in violence when the patient was watched. She was unable to use a knife or fork, and her food was cut up for her. The patient was, however, able to eat with a spoon, though with considerable difficulty. She was able to drink from a tumbler by holding it with both hands, but was unable to wash her face or dress herself. She never spoke about friends or relations. She would only eat at meal times, and if food were presented to her between meals, she would throw it at the head of the person offering it. She would not permit the clergyman or visiting ladies to converse with her, and would attempt to throw things at them or curse them. The patient could walk without assistance by taking a step, resting for a short time, and then taking another. As a rule, however, she helped herself by holding on to the beds. Upon several occasions, however, I have seen her walk rapidly through the wards with an irregular gait, the arms swinging violently in all directions, and the head tossed from side to side by the violence of the choreiform movements in the muscles of the neck. She would rarely leave her chair except to go to the closet. Even while sitting in her chair, the body and limbs underwent violent contortions so that she retained her position with some difficulty. During sleep the movements ceased entirely. The patient would never take medicine and would not allow anybody to go near her except another patient in the same ward. During the last month she had been gradually failing, becoming weak and anæmic. Upon several occasions she fell to the floor, and, about a month ago, sustained a severe contusion of the right side, face and eye. She never complained since her admission to the hospital, but groaned occasionally during the last week. The patient has been failing very rapidly for the last two or three days, but would not take to her bed; was found dead in her chair, November 8, 1878.

Autopsy.—Atrophy of the ascending frontal and parietal convolutions upon either side; sclerosis of the medullary substance of both hemispheres (middle third); descending degeneration of the crura, pons, medulla, and cord; cardiac hypertrophy; cirrhosis of the liver; chronic diffuse nephritis.

The post-mortem was held forty-six hours after death. Body of medium size, emaciated, rigor mortis present; contusion over right frontal bone, slight œdema of the eyelids.

Head.—*Calvarium* thin and the bones easily cut with a knife. *Dura mater* shows a pachymeningitis of the falx cerebri; the *sinuses* normal. *Pia mater* normal with the exception of marked œdema, which is especially evident over the paracentral lobule on each side, corresponding to the atrophy of the underlying convolutions. The *vessels* at the base of the brain show thickening of their walls (in places) from atheroma and a deposit of calcareous plates. The lumen of the affected vessels is moderately narrowed, but all are pervious, so far as examined.

The pia mater can be everywhere normally stripped from the convolutions. *Convolutions.*—The ascending frontal and parietal convolutions upon either side are moderately atrophied from their lower third up to the longitudinal fissure. The atrophy is most marked on the right side, at the situation of the paracentral lobule. Externally, the color of the atrophied gyri shows nothing different from that of the unaffected convolutions, but, upon vertical section their outer third presents a bluish gelatinous appearance, and the inner two-thirds are of a deeper yellow and more granular appearance than elsewhere. The affected convolutions are not abnormally softened.

Lateral ventricles.—The ependyma is thickened and presents granulations upon its surface; upon the right side over the corpus striatum this thickening is most marked and corresponds to evident atrophy of the interventricular nucleus. The third and fourth ventricles show nothing abnormal. There is no marked dilatation of, or increase of fluid in the ventricles. On section of the brain substance, the white matter is found to be sclerosed in the middle third of each hemisphere and extending somewhat beyond this boundary anteriorly. The sclerotic portions are tough and leathery to the feel, and have a pinkish blue or mother-of-pearl color. There is a symmetrical spot of white softening in the middle of the outer segment of each lenticular nucleus. With the exception of this softening, and of the previously mentioned atrophy of the right interventricular nucleus, the basal ganglia are normal. The crura cerebri are about equal in size; upon transverse section, each shows central atrophy, which is most marked on the right side, with slightly yellowish discoloration of the white fibres bordering on the locus niger, and transformation of the normal black pigmentation of the latter to a dull yellowish brown.

Pons varolii.—Upon inspection, the lateral halves appear to be of equal size, but on transverse section the bundles of longitudinal fibres of the right side are scarcely more than half the size of those on the left.

Medulla oblongata.—There are no marked changes to the naked eye.

Spinal cord.—This exhibits a descending degeneration which, to gross appearances, disappears in the lumbar region, and is throughout most marked in the left half of the cord. The meninges of the cord are normal.

Thorax.—The sternum fractures upon the application of the slightest force. Several ribs are fractured and, at the corresponding portions of the costal pleura there are evidences of localized pleurisy and subpleural hemorrhages. *Lungs* exhibit emphysema and bronchitis.

Heart.—Hypertrophied, especially the left ventricle.

Aorta.—Dilated and atheromatous, with calcareous plates.

Abdomen.—*Liver*, markedly cirrhotic. *Kidneys.*—Atrophied, the cortex fatty and filled with cysts (chronic diffuse nephritis).

The above mentioned cases will suffice to furnish an idea of the variety of anatomical lesions which have been found present in chorea.

We must not forget, however, that a considerable number of cases have been reported by competent and careful observers, in which no material lesions of the nervous system were found either upon macroscopical or microscopical examination.

CHAPTER IV.

PATHOLOGY.

EVEN at the present day, opinions vary as to the cerebral or spinal origin of chorea. The chief arguments in favor of the spinal character of the affection have been advanced by French physiologists, especially Chauveau, Longet, Carville and Bert, Legros and Onimus.

Chauveau [1] experimented upon dogs who were suffering from general choreiform movements, by dividing the spinal cord immediately below the medulla oblongata. He found that the chorea nevertheless persisted until the death of the animal with the same degree of severity. If the spinal cord was divided, however, through the dorsal region, the choreiform movements immediately ceased in the tail and posterior limbs, but remained unaffected in the anterior limbs.

The experiments of Longet, Carville and Bert, were merely confirmatory of the results obtained by Chauveau.

Legros and Onimus [2] made more extended investigations concerning this question. Like the authors previously mentioned, they also found that section of the spinal cord immediately below the medulla oblongata did not diminish the violence of the choreiform movements, if artificial respiration were maintained. If the spinal cord was laid bare by removing the vertebral arches, and the posterior columns of the cord were irritated with the end of a scalpel, the choreiform movements were exaggerated to an enormous extent. If the cord were cooled by means of a current of air, the movements disappeared when the previous temperature of the cord was restored by pouring warm water upon it. The movements were uninfluenced by excision of the posterior roots of the spinal nerves, but were diminished by a partial incision through the posterior white columns and posterior horns of gray matter. When a deep incision was made through these regions, the choreiform movements were entirely abolished.

Legros and Onimus think they are justified in concluding from these experiments that the morbid process in chorea is situated in the cells of the posterior gray horns, or in the fibres which connect these cells with the large motor-cells of the anterior horns.

These experiments appear to us neither to prove nor disprove the spinal character of chorea in the human subject. Apart from the general suspicion which justly attaches to the drawing of conclusions concerning functional disorders in man from the results of experiments in animals, it is quite positive that the chorea of dogs is an entirely different disease from the similar affection in man. This, of course, invalidates all conclusions which are drawn concerning the latter from the morbid appearances presented in the former.

A review of the pathological anatomy of the affection, as described in

---

[1] Arch. gén. de méd., 1866.

[2] Comptes rend., LX., 1870.

the last chapter, teaches us that the lesions of chorea have been found both in the brain and spinal cord, and, as Elischer's results appear to indicate, in the peripheral nervous system as well. In numerous cases, on the other hand, the entire nervous system has been found intact. It is evident, therefore, that pathological anatomy cannot thoroughly explain the pathology of the affection, and we must resort for further light to the clinical history of the disease. In fact, it appears to us that undue importance has been hitherto attached to anatomical investigations in the study of the pathology of functional diseases of the nervous system, and too little to the clinical symptoms of these diseases and to their etiological relations.

The rapid and brilliant development of pathological anatomy within the last twenty-five years has laid such a strong hold upon the minds of medical men and upon their habits of thought, that they are apt to rush to the conclusion that the presence of a certain anatomical lesion in any disease is conclusive evidence that such lesion constitutes the real cause of the affection, and to draw a hasty generalization from very meagre data. In no department of medicine is this more marked than in diseases of the nervous system, and not a small proportion of the investigations now being carried on in this field will result in overturning certain of the theories to which credence has been hastily and incautiously given.

In the first place, the weight of evidence appears to us to be overwhelmingly in favor of the cerebral character of the affection. The fact that hemichorea is sometimes combined with hemianesthesia, and that the latter symptom is always observed upon the same side as the choreiform movements, is proof positive that the lesion is cerebral in its origin. If it were of a spinal nature, the decussation of the sensory fibres in the cord would cause the appearance of the sensory symptoms upon the side opposite to the motor disturbances.

Another important argument in this particular is the fact that mental disturbances are so common in the affection under consideration. In all the cases which have come under my observation, there was at least some change in the disposition of the patient, consisting of irritability, deficient memory, or obstinacy. As we have further shown in the chapter on the clinical history of the disease, more marked mental disturbances, advancing even to insanity, are not very infrequent.

Chorea, also, may occur in one member of a family, others of whom present epilepsy or insanity, or the chorea may alternate in the same patient with either of these affections, *i. e.*, the hereditary neuropathic tendency may, in one case, involve one portion of the brain, giving rise to insanity, in a second, affect another part causing epilepsy, and, in a third, produce chorea.

Furthermore, chorea may develop during the course of well-defined cerebral diseases, such as hemorrhages, tumors, meningitis. In the rare cases of prænemiplegic chorea, the choreiform movements appear as the precursors of the cerebral affection, and are undoubtedly due to the initial development of the cerebral lesion.

Finally, movements of the facial muscles, which are supplied by cranial nerves, are noticed in the vast majority of cases of chorea, a symptom which is inexplicable on the theory of the spinal origin of the disease.

But, although there is good ground for the belief that chorea is a cerebral affection, the theories with regard to the nature and position of the lesion are extremely various.

Sée, struck by the frequent coincidence of chorea and rheumatism or

endocarditis, regarded both processes as very closely related, but gave no very satisfactory account of the connection between them.

Some years later, Roger advocated the doctrine that chorea, rheumatism, and endocarditis are so many manifestations of one diathesis, the difference in the clinical history of the three affections depending merely upon the organ affected. But this sweeping statement is amply disproved by the results of the statistics which we have collected in the chapter on etiology, and also by our own experience. Thus, in upward of ninety cases which have come under my observation, in only three were there any evidences of rheumatism.

Nevertheless, the results of autopsical examinations have at least proven the frequent coexistence of chorea and endocarditis. Kirkes first propounded the "embolismic" theory of the connection existing between these two affections. He considered the theory of a rheumatic diathesis as the causal factor of rheumatic arthritis, endocarditis, and chorea unsatisfactory, because the two latter diseases are often associated, although no trace of previous rheumatism can be discovered. He regards it as very probable that chorea is due to removal of some of the fibrinous particles from the valves of the heart and to the disturbances created in the nerve-centres by the impaction of such particles in the cerebral vessels. At a later period Broadbent, in an article read before the London Medical Society, narrowed down the doctrine more closely, by stating that the chief cause of chorea is capillary embolism of the basal ganglia (corpus striatum and optic thalamus) and of the structures in their immediate vicinity. This theory was also strengthened by the investigations of numerous English observers, notably Hughlings Jackson, Tuckwell, and Ogle.

W. Howship Dickinson, although he acknowledges the close relationship of chorea and endocarditis, thinks that the latter affection is a secondary process, due to the choreiform movements of the papillary muscles of the heart. As we have previously stated in our remarks on the clinical history of the affection, it is extremely problematical whether the muscular tissue of the heart is ever the seat of choreiform contractions. But, even if this condition could be proven, we are unable to understand in what manner it would lead to inflammation of the endocardium.

Although the embolismic theory of chorea is very plausible and fascinating, there are, in our opinion, weighty objections to be urged against its acceptance.

In the first place, in ordinary cerebral embolism, the embolismic particles, from causes which it is not necessary to enter into in this place, are usually distributed to the vessels on the left side of the brain.

Ottomar Gelpke[1] denies the truth of this statement to the extent to which it is generally believed, and finds from an analysis of 131 cases of cerebral embolism that sixty-four occurred upon the left side, fifty-four on the right, and thirteen on both sides, thus rendering the proportion on the two sides almost equal. Bertin,[2] however, found thirty-six cases of ebmolism on the left side of the brain, and only nine on the right side. Gerhardt[3] observed the embolus thirty-five times in the left middle cerebral artery, and sixteen times in the right. Although I have been unable to obtain any statistics on the subject, I am positive that, among a

---

[1] Arch. d. Heilk. 1875. [2] Étude critique de l'embolie.
[3] Wuerzb. med. Zschr. 1863.

large number of cases of cerebral embolism which I have had the opportunity of observing post-mortem in the dead-house of Bellevue Hospital, very few cases have occurred upon the right side of the brain. Dr. Janeway has also called my attention to the fact that the ordinary statistics with regard to the relative frequency of right and left cerebral embolism are unreliable, because, from the comparative rarity of right-sided embolism cases of this nature are apt to be published, while left-sided embolism is not considered, as a rule, worthy of publication.

In the second place, the clinical history of chorea differs from that of cerebral embolism. The paralyses which occur in the former, almost invariably recover very rapidly, and do not come on as suddenly as those dependent upon embolism. While a choreal paralysis may recover completely within a couple of weeks, a case of equally severe paralysis from cerebral embolism will occupy several months in recovery, and even then, as a rule, some paresis of the affected limbs is still perceptible.

Furthermore, while true aphasia not infrequently occurs in cerebral embolism, this symptom does not develop in chorea. The disorder of speech which occurs so frequently in the latter disease is due to an extension of the affection to the muscles of phonation, and is not of a cerebral nature, properly speaking, in the same sense that aphasia is.

Finally, the chief argument, perhaps, against the acceptance of the embolismic theory of chorea lies in the fact that the lesion is found so seldom by pathologists although search has been carefully made.

The theory of capillary embolism restricts the lesion of chorea to the basal ganglia, since the collateral circulation is so free in the cortex of the brain that plugging of a few capillaries would produce no evil effects upon the nutrition of the nerve-structures in that locality. But a lesion of the motor and sensory basal ganglia will not produce the mental disturbances which, as we again insist, form such an integral part of the clinical history of the affection.

Somewhat similar to the embolismic theory of chorea is Bastian's theory of capillary thrombosis, which he formulates as follows: chorea is due to an altered and often anæmic state of the blood which chiefly acts upon the corpora striata and surrounding parts, causing the tissue elements to become "irritated;" congestion then develops as a necessary consequence. If the irritation continues for a certain length of time, the disturbed action outside of the vessels is communicated to the tissue elements within them, the white corpuscles therefore begin to adhere to the walls of the small vessels, so that partial obstructions may be produced, which are, perhaps, afterward rendered complete by the separation of fibrine or allied products. In exceptional cases (observed by Aitken and Tuckwell), small foci of softening result from these processes.

Apart from the slight amount of support which this theory receives from post-mortem investigations, the same objections apply to it as were advanced in opposition to the doctrine of the embolismic origin of chorea. Furthermore, neither of these theories is capable of explaining the numerous class of cases in which the disease evidently results from fright, from reflex causes, or from imitation.

It seems to us that very little aid can be derived from pathological anatomy in determining the situation of the lesion in chorea. The vast majority of patients recover from this disease without presenting any severe symptoms, and it is therefore highly improbable that a serious anatomical brain lesion was present. As shown in the chapter on pathological anatomy, chorea may accompany various cerebral lesions, but these

should then be regarded rather as complications than as essential features of the disease.

The symptomatology of chorea may be broadly considered as consisting of mental disturbances (irritability, loss of memory, perhaps mania, etc.), and motor disorders (muscular twitchings, certain amount of paresis).

These symptoms could be readily explained by a lesion of the cerebral cortex in the neighborhood of Hitzig's motor centres and the adjacent parts of the frontal convolutions. Like all the functional neuroses, chorea is an evidence of low tone of the nervous system, and we may accordingly regard the cortical disturbance either as the result of anæmia in the parts affected, or of malnutrition or exhaustion of the ganglion cells in the convolutions. As a matter of course we can offer no demonstrable proof of the correctness of this view, but it appears to tally best with the results of clinical observation.

To our thinking, no theory can be correct which does not explain the general run of cases, and not alone those which are attended with exceptional symptoms or terminate fatally. The theory of malnutrition of the cortical ganglion cells, either as the result of anæmia or of a direct effect upon the nerve-cells, such as is known to occur at times as the result of fright, appears to us to be capable of explaining the large majority of cases of the disease.

In this connection we desire to call attention to the views expressed by Sturges[1] who has written some admirable articles on the disease under consideration. This author believes, "that the pathology of an affection like chorea is to be sought in the natural endowments of the individual; that it comes to be a child's disease because those elements out of which it is evolved are especially prominent in early life; that childhood not only *predisposes* to chorea, but has also the material which is efficient to produce it." He points out the identity of the movements of restlessness in a shy child when embarrassed, with those observed in chorea, and believes that the latter may grow directly out of the former as the result of fright, pain, etc.

---

[1] Med. Times and Gazette, April 28, 1877.

CHAPTER V.

DIAGNOSIS AND PROGNOSIS.

THE clinical history of chorea minor is so sharply defined that it is very rarely mistaken for other diseases. In children, as well as in adults, care must be taken to differentiate it from post-hemiplegic chorea. An inquiry into the previous history of the patient will soon dispel any doubts which may exist with regard to the diagnosis. The post-hemiplegic affection is unilateral, and, as a rule, when the movements have begun in the first five or six years of life, the limbs of the affected side are atrophied in all directions; this does not hold good, however, with regard to post-hemiplegic chorea of adult life. The movements in the post-hemiplegic affection generally subside entirely, when the patient is not performing any voluntary movements, and are usually not so rapid or sudden as those of ordinary chorea. After the disease has lasted for a long time, a mere examination of the patient may be insufficient to determine the true nature of the affection, as the original loss of power in the limbs may have disappeared, leaving only the choreic movements.

The nature of pre-hemiplegic chorea (which is an extremely rare affection) will only be cleared up by the further history of the case, unless apoplectic symptoms develop from the onset and accompany the choreiform movements. These movements may continue, however, for several days before the development of hemiplegia, etc., warns us of the true character of the affection with which we have to deal.

In rare instances, it will become necessary to differentiate chorea from cerebro-spinal sclerosis. As a rule this can be readily done from a consideration of the clinical history. In multiple sclerosis, the patients suffer usually from marked sensory disturbances (pain, anæsthesia in the limbs), from paresis or contracture of muscles, bladder symptoms, pupillary phenomena, cerebral disturbances. Occasionally, however, the motor disorders constitute the chief symptom (perhaps for a certain length of time the only discoverable one), and we must therefore directly compare the motor phenomena of chorea and sclerosis.

As a rule, the movements of chorea develop independently of any voluntary effort on the part of the patient; the latter may be sitting perfectly quiet when suddenly the fingers twitch, the muscles of the face contract, or the tongue is protruded from the mouth. In cerebro-spinal sclerosis, however, the irregular movements are not evident unless the patient makes a voluntary effort[1] and the greater the will-power

---

[1] Charcot was the first to call attention to this point, and lays it down as an absolute rule. That exceptions do occur, is proven by a case which I observed in Dr. Janeway's practice, and in which the autopsy showed the nature of the affection.

exerted the more violent do the movements become. Another point of contrast between the two affections consists in the fact that, in cerebro-spinal sclerosis, the general direction of the intended movement is maintained throughout, while in correspondingly severe cases of chorea, the patient deviates in all directions from his intended course. Thus, a patient suffering from multiple sclerosis will be able to carry a spoon or glass to his mouth (though some of the contents will be spilled), but in a severe case of chorea, the individual will strike the object against his ear or nose; as soon as the spoon is about to enter his mouth, a sudden jerk of the arm will draw it forcibly away. The difference between the two classes of movements appears to me to be best formulated by the statement that, in chorea, volition is disordered at the time of its development in the cerebral cortex (in which I presume the lesion is situated), while in cerebro-spinal sclerosis, the nerve-force becomes interfered with, as Charcot expresses it, in its passage through the affected portions of the spinal cord.

Only the grossest carelessness could give rise to the mistake of regarding the movements of paralysis agitans as choreiform in their nature. The former consist of tremor of a limb, as a whole, the excursions of the muscles being very short and following one another with great regularity. Until the disease has lasted for a very long time, the movements can always be quieted by a strong effort of the will, thus differing at the same time from the twitchings of chorea and of multiple cerebro-spinal sclerosis, for which this affection is also sometimes mistaken. The clinical history of paralysis agitans and chorea is entirely different. The former is a disease of old age (exceptionally it occurs in middle life); it is almost impossible to check the disease, the movements rarely involve the head (the movements of the head are communicated from the trunk), and the affected muscles usually present a certain amount of rigidity—phenomena which are all entirely distinct from those observed in chorea.

The *prognosis* of chorea, as regards recovery from any single attack, is ordinarily good. As a rule, the disease, after it has reached its culmination, begins slowly to decline, and most cases have run their course in a period varying from two to four months. But a considerable percentage of the cases suffer from relapses, and we not infrequently find, upon inquiring into this point, that the patient has had an attack every spring for several years in succession, or perhaps, in alternate years.

Sometimes the patients have two attacks in one year, each lasting several months. In these cases, as well as in all others in which the disease has lasted over six months, there is danger that the chorea may continue for years, and even until the end of life. I have at present a case of this kind under observation, in which the choreic movements have continued with undiminished severity for nearly a year, and in which the mental faculties are gradually failing, so that I apprehend the development of complete dementia.

The prognosis, as regards a fatal termination, is very good, and not a single case of death (with the exception of the patient mentioned on page 27, in whom cerebral sclerosis was found after death) has come under my notice. Cases are reported, however, in which exhaustion and death supervened on account of the extreme violence of the muscular twitchings, the loss of sleep caused thereby, the lack of nutrition, and sometimes exhaustion from mania. Such instances are, however, quite infre*quent,* especially in this country. As a rule, death is due to the pres-

ence of some intercurrent affection, such as cerebral hemorrhage, endocarditis, etc.

The psychical complications of chorea present a very favorable prognosis as regards recovery from the mental affection, though we must admit that not an inconsiderable proportion of the fatal cases have been attended with mania. As a rule, however, the patient entirely recovers from the maniacal disorder, and the improvement keeps pace with the diminution in the choreic movements, although the mental disturbance may sometimes last several months after the former have disappeared.

CHAPTER VI.

TREATMENT.

THE first step in the treatment of chorea is to take the children (the disease, as we have seen, generally occurs in childhood) away from school. This is beneficial in several ways. In the first place, the memory is usually impaired to a greater or less extent in this affection, and the little patients are not so bright and intelligent as in their normal condition. The consequence is that they must make unusual efforts to retain their standing in the class, and this strain, reacting upon an already irritable brain, serves to aggravate the existing functional disorder. Furthermore, the patient's classmates, with the well-known inconsiderateness of childhood, are apt to deride and jeer the little sufferer on account of the grotesque character of the muscular movements to which he is subject, and the shame and anger which are produced in this manner will give rise to the same bad effects as those due to mental overwork.

Shall the patient be allowed to run at will in the open air? We are aware that this is the usual advice given by the physician, but we are not by any means convinced of its utility. Children are apt to engage in sports with all the earnestness of a serious pursuit, and usually become thoroughly tired out in consequence. This is especially true with regard to choreic children, whose muscular energy always becomes diminished during the course of the disease. It is a well-known fact, however, that rest tends to diminish the violence of choreic twitchings, and for this reason, although I favor out-of-door life for the patient, I discountenance any rough sports, and prefer that he should take short walks, carriage-rides, etc.

For a similar reason, also, the patient should be allowed to indulge in sufficient sleep. Although the choreic movements usually begin as soon as the patients wake, or within a few minutes afterward, they are not so severe as they are after the lapse of an hour or two. Sleep is frequently disturbed in this affection, and as this is sometimes due to the fact that the patients are worn out from the constant muscular movements, it is advisable to give the patients a small quantity of ale or porter before retiring. When this simple measure proves unsuccessful in procuring a sound sleep, we are warranted in giving from five to ten grain doses of hydrate of chloral at night. We will often find that after several nights' good rest has been secured in this manner, the twitchings rapidly diminish in severity. It is not advisable, however, to continue the use of chloral for any length of time, for we find that in children especially it is apt to give rise to bad after-effects, and is a very potent factor in producing and maintaining an anæmic condition.

Some of the recommendations presented by Dr. Sturges with regard to the treatment of chorea are so excellent that we shall make a short abstract from one of his lectures.

"Chorea is aggravated by emotion and close inspection; it ameliorates

with mental and bodily repose and preoccupation; it ceases altogether in sleep and during intervals of musing. Any method of treatment which places the child under obvious surveillance, and thus makes it attentive to itself, must tend to aggravate the complaint and aid in its development. . . . . To rest the overworked and tired limbs, to secure a large measure of sleep; to make the time pass evenly, yet without the weariness of monotony; to save the voluntary muscles the mortification of failure by anticipating the child's wants; these, as I believe, are the most serviceable duties which can be rendered at the outset of chorea. Yet they must be done without ostentation, and without the child perceiving that he is being tended and watched and treated as one sick."

The patients should be allowed a free, generous diet, and no restriction need be made with regard to the character of the food, except under special circumstances. As in all neuroses, which are evidence of a low condition of nervous tone, it is well to introduce as much fat as possible into the food, and with this end in view we may administer milk and cod-liver oil. In the majority of cases the appetite is poor and usually capricious, and we may then prescribe some of the simple bitters (calumba, quassia, gentian).

A considerable number of the patients are anæmic from the beginning, while a much larger number become so in consequence of the character of the disease. In these cases, mild ferruginous tonics are indicated, such as dialyzed iron or carbonate of iron, or the tincture of the chloride of iron, which is preferable when the digestive organs are in good condition. It is unnecessary, however, to administer iron as a routine matter of treatment, as is so frequently done, under the impression that iron possesses a certain specific influence in the treatment of the disease.

The medicinal treatment of chorea is extremely unsatisfactory. Gray and Tuckwell found that the average duration of thirty-eight cases, which were treated on the expectant plan, was nine weeks and six days. The average duration when treated with arsenic in gradually increasing doses, according to Begbie's plan, was ten to eleven weeks. Sée gave sixty-nine days as the average duration of 117 cases treated with various medicines, and it is a curious coincidence that this agrees exactly with the duration of Gray's and Tuckwell's cases. The majority of observers agree that recovery within two to three months constitutes a good result. My usual plan of treatment is to put the patient on three to five drop doses of Fowler's solution (three times a day, immediately after meals, in a little water), and increase this amount by one drop at a dose until some of the toxic effects become evident (nausea, sometimes vomiting or looseness of the bowels, slight œdema of the eyelids, perhaps pitting over the tibiæ). The drug is then discontinued for a few days until these symptoms have subsided (an alkaline drink such as Vichy water will accelerate their disappearance), and it is then again administered in doses slightly smaller than those which sufficed to produce the above-mentioned toxic effects. If it produces any good results, the remedy may be continued in this manner for a period of six or eight weeks. I have found that although this plan of treatment will not cut short the disease, the movements will become very much milder within ten days or two weeks, but that some amount of motor disturbance will persist until the disease has run its natural course (two to three months).

When the muscular twitchings are of an extremely violent character, interfere with sleep, and threaten to produce serious prostration, I have

obtained good results from the use of a mixture of bromide of potassium and hydrate of chloral. The dosage depends upon the severity of the individual case, but the largest amount should be given at night. Prolonged warm baths (fifteen minutes to half an hour), are also useful under these circumstances. The movements are sometimes so violent that it becomes necessary to tie the patient in bed in order to prevent him from doing injury to himself. It is well in all cases of such severity to keep the patient in bed continuously until the excessive violence of the movements subside. The condition of the integument should be carefully examined from day to day in order to detect the development of excoriations, which are liable to be produced from the violent friction of the parts against surrounding objects.

During the past year I have made quite extensive use of inhalations of nitrite of amyl, beginning with two-drop doses three times a day and gradually increasing to six or seven drop doses. It has seemed to me that its effects may be compared with those of Fowler's solution, *i. e.*, it produces considerable improvement within one or two weeks, but the disease then runs its usual course.

It is well to bear in mind, in treating chorea, that the patients rarely fall into our hands until the disease has lasted for a longer or shorter period, and that part of the effect, which we attribute to our remedies, may be due to Dame Nature.

Strychnia has been highly recommended by many writers. We must exercise caution in its administration to children, as they not infrequently present a peculiar susceptibility to its influence. A child of eight or nine years should not receive more than one one-hundredth of a grain at a dose in the beginning, and this quantity may then be gradually and cautiously increased. In my hands the drug has proven almost useless except as a nerve-tonic.

Within the last few years Bouchut has introduced eserine as a remedy in this disease, and claims truly wonderful results in a very large number of cases. It was administered in doses of about one-sixtieth of a grain, either by the mouth or as a hypodermic injection, and Bouchut claims that the majority of cases recovered within two weeks. These remarkable results have, however, not been obtained by other observers, and its administration is sometimes attended with such disagreeable effects (vomiting, prostration, etc.) that I have entirely refrained from using it.

Curare has been successfully employed in a few cases of chorea of old age, which is usually regarded as incurable. This is given in doses of one-tenth of a grain in the beginning, and of course by means of hypodermic injection, since it is well known that when this drug is administered by the mouth, it is eliminated so rapidly by the kidneys that no effects upon the nervous system are produced. If good results are not obtained within a week after beginning its use, it would be unwise to continue its administration.

Numerous other drugs have been employed in the treatment of this disease, but it is unnecessary to refer to them, as none of them have stood the test of experience.

Ottomar Rosenbach has advised the employment of the constant galvanic current and of counter-irritation applied to the tender spots which are found along the spinal column. I resorted to this plan of treatment in three cases, but without obtaining the slightest improvement. Numerous observers have employed the constant current (to the spine) in treating chorea, but the large majority concur in the opinion that it is

either entirely useless, or that its temporary musculo-sedative effects soon disappear.

In the severe cases which are attended with maniacal excitement and extremely violent muscular contortions, it is advisable to put the patient in a straight jacket,[1] administer large doses of bromide of potassium and chloral, and endeavor, by every means in our power, to feed the patient. When this cannot be done by the mouth or through the stomach-tube, nutritious enemata should be at once resorted to. In administering hydrate of chloral in such cases, we should bear in mind that the continual movements of the body tend to antagonize the effects of the drug, and that large doses (ten to thirty grains, or even more) should be given. When the choreic twitchings can be controlled in no other manner, we may be compelled to resort to inhalations of chloroform.

[1] This is advisable, not on account of the mania, but merely to moderate the excessive muscular twitchings, the production of which is a source of fresh contortions.

EPILEPSY.

CHAPTER I.

CLINICAL HISTORY.

THIS dread disease has been known since the earliest periods in the history of medicine, and has always attracted the attention alike of the profession and of the laity. The peculiar character of the symptoms, and the usually incurable nature of the malady, caused it to be looked upon as an evidence of demoniac possession. The terms applied to it by the lower classes, in some countries, indicate that this view obtains even at the present. The conception which was formed of the disease remained unchanged for centuries, and it is only within comparatively recent times that our views concerning the scope and boundaries of this affection have undergone a radical change and amplification. It was formerly regarded as a paroxysmal disease, in which the paroxysms consisted essentially of unconsciousness and general convulsive movements, but, at the present time this combination of symptoms is only looked upon as characteristic of one form of epilepsy, viz., the *grand mal.* In addition to this variety there are cases in which the loss of consciousness is the only symptom, the convulsive movements being entirely absent. These two varieties are not, however, sharply separated from one another, and there are forms which are intermediate between the two, *i. e.*, there are cases in which the unconsciousness is combined with partial convulsions. There is another variety in which the attacks are characterized by peculiar groups of actions performed while the patient is in an unconscious or partly unconscious condition. Finally, a new group has been placed under this head by Griesinger, who applied the term "epileptoid states" to the manifestations in question.

GRAND MAL.

We shall first enter upon the consideration of epilepsia gravior, or the *grand mal.* The history of the affection embraces two parts, viz., the paroxysm itself and the interparoxysmal period. The attack frequently begins without any warning. The patient, while engaged in his ordinary occupations, suddenly loses consciousness, his face grows pale, and at times he utters a peculiar inarticulate cry. He then falls as if struck by a heavy blow, and the convulsive phenomena immediately become apparent. The stage of tonic convulsions now begins. The spasms

appear first in the muscles of the face or the small muscles of the hand, and then rapidly spread throughout the entire body. Slight twitchings are observed in the muscles inserted into the angles of the mouth; the eyes become fixed and are usually drawn to one side; the muscles of the neck contract, and frequently draw it to the side toward which the eyes are turned; the muscles of the hand contract, and draw the thumb firmly into the palm, and the fingers are clenched. At the same time the muscles of respiration, including the diaphragm, are similarly affected and impede the act of breathing; this is immediately followed by powerful tonic contractions throughout all the muscles of the body. During the entire stage, which only lasts from a few seconds to a couple of minutes, the body does not move from the position in which it has fallen. These contractions may be so powerful that the head is drawn forcibly backward, and the entire body is in a condition of slight opisthotonos, or, if the contractions are stronger on one side, as they frequently are, in a condition of emprosthotonos. The tonic spasm of the muscles then begins to relax, and clonic convulsions make their appearance, at first mild and localized and then growing more diffuse and violent, until, in a very short time, the whole body is in a continual state of violent muscular contraction and relaxation. The face, which had begun to grow dark during the latter part of the tonic stage, now becomes dusky and turgid, the eyes stare, and the features are disfigured by terrible grimaces and contortions. During this period, the tongue, which has been protruded by the violence of the muscular action, is frequently caught between the rapidly closing jaws. After a variable period (usually from two to five minutes) this condition subsides. The clonic contractions sometimes cease quite suddenly, but usually they grow milder gradually, the dusky hue of the face and body begins to disappear, and the patient then lays perfectly quiet. In some cases, he immediately rouses from his comatose condition, opens his eyes, and looks around him with a stupid, frightened air, mutters, perhaps, some inarticulate words, and then rapidly lapses into his previous condition. Generally, however, he falls into a deep, lethargic sleep, from which he may be roused into consciousness, and wakes up after a period varying from a few minutes to several hours; the epileptic coma has even been known to continue forty-eight hours. The patient usually wakes feeling dull and heavy, complains of headache, and has a tired aching feeling in the muscles, as if he had been engaged in some very heavy work.

The attacks do not always begin as suddenly as we have described, but in some cases prodromata are experienced, so that the patients can always foretell the occurrence of a convulsion. Opinions vary with regard to the frequeney of these symptoms. Some authorities, including Romberg and Sieveking, state that they have met them in one-half of all their cases, others have observed them with much less frequency.

The character of these prodromic symptoms is manifold; they are usually classified into the remote and immediate. The former may last for one, two, or even three days, and are often manifested by a change in the disposition of the patient. If he has previously been in his usual condition of cheerfulness, he now becomes gloomy and irritable, and is liable to causeless attacks of anger; or, perhaps, a sullen state gives way to a more cheerful demeanor. If those surrounding the patient are attentive and observant, they are generally able to tell, from this change of disposition, that a convulsion is impending. Reynolds has also noticed a peculiar *remote* prodroma, consisting of a duskiness of the skin, especially

affecting the face and neck, this symptom being observed from four to twelve hours before the onset of the attack.

In one case under my observation, the patient, for twenty-four to thirty-six hours before the fit, has prodromata which consist of palpitation of the heart, and a feeling of heat arising from the præcordium, together with pain and a feeling of distention in the abdomen; she, also, has a flushed face and headache during this period.

In another case, the patient feels dull, and complains of pain in the frontal region for about a day before the fit; during this time she also has a watery diarrhœa (ten to twelve passages) although the bowels are perfectly regular at other times. I also have under my care a patient in whom these abdominal prodromata are very well marked (I may mention here that these three patients are females). The patient in question is forty-eight years old, and has had epileptic attacks during the last ten years. For twenty-four hours before the attack begins, she suffers from uncontrollable vomiting and diarrhœa, so that she is compelled to keep to her bed. At times this is accompanied by intense pain in either forehead, or by exquisite hyperæsthesia in localized spots in various parts of the limbs. Immediately before the convulsion begins, the patient has a sensation as if a large ball were situated in the anus, this appears to move up the rectum, and then passes to the lumbar region of the spine, along which it mounts; she then loses consciousness and has a severe convulsion.

An extremely rare prodromal symptom was observed by Kuethe.[1] He found that the individual in question manifested agraphia prior to one of his attacks. The patient, who was a book-keeper, was attacked with a convulsion while engaged in writing in his account books. An examination of these books showed that before the attack began, the patient made wrong entries, repeated certain syllables several times in succession, and introduced words which had no bearing on the accounts.

The immediate prodromata are usually known under the term aura, and are much more variable in their appearance. They may be classified as psychical, motor, sensory, and vaso-motor.

At times the patients feel giddy immediately before the attack, and this feeling sometimes lasts sufficiently long to allow them to secure a safe position before the convulsion occurs. At other times they experience a peculiar feeling in the head, which only lasts for a few seconds, and which very intelligent patients have told me is perfectly indescribable. Or, the patient may be in a peculiar state of excitement, and evince great loquacity for a few seconds or minutes before the fit.

The sensory aura are more variable than the psychical ones. Sometimes the convulsion is preceded by peculiar hallucinations. Watson relates that Dr. Gregory, of Edinburgh, was assured by a patient of undoubted veracity, "that always when he had a fit of epilepsy approaching he fancied that he saw a little old woman in a red cloak, who came up to him and struck him a blow on the head, and then he immediately lost all recollection and fell down."

The visual prodromata may also merely consist of sensations of light, in which the red color appears to predominate. An affection of the acoustic, olfactory and gustatory nerves is also sometimes met with. Thus, the patient may hear a buzzing or roaring noise in the ears, ringing of bells, may smell a peculiar stench, or have a curious taste in the mouth. In one case, a patient of mine, before having the fits thought he heard a

[1] Arch. f. Psych. 1879, pp. 257-260.

buzzing noise above him, which came down, entered his head, and then went to his stomach, after which he lost consciousness.

The nerves of general sensibility are affected as well as those of special sense. In fact, the term aura, which refers to the sensation of a breath, strictly applies only to sensory prodromata. The latter may consist of painful sensations, starting from some part of the trunk or limbs and rising rapidly to the head, or of a sensation in the head as if something had cracked there, etc. Instead of pain there may be a sensation of numbness, or even true anæsthesia. Nothnagel relates that in some of his patients, in whom this phenomenon lasted ten minutes or longer, he was able to detect a palpable diminution of sensitiveness to the prick of a pin or to changes of temperature. A very frequent sensory aura consists of a feeling of pain in the epigastrium or lower down in the abdomen, which rapidly rises to the throat; in other cases, the patients describe the sensation as of a jumping or jerking character, although no movements can be observed. As soon as the sensation reaches the throat or head, unconsciousness supervenes. I have already referred to the case in which the patient felt as if a large ball started from the anus and ran up the rectum; it then appeared to pass up to the back, after which the fit immediately developed.

The motor aura consist either of muscular contractions or paralysis, which usually affect one limb or even the whole side of the body. The paralytic aura is extremely rare, and has never come under my notice; some authorities even doubt its existence. The motor aura is readily understood from a few illustrative cases. In one typical example the patient, a young man, while strapping his valise very tightly, noticed that his right hand became violently extended and was then rapidly flexed; he turned to walk to his father, who was standing at some distance from him, and while doing so, the clonic contractions spread to the forearm and then to the arm; he then lost consciousness, and the convulsion developed. In another case, a man, who had received an injury to the lower and outer part of the left leg several months previously, observed some twitchings in this part which then spread to the thigh, the left side, and finally the left arm, after which he immediately became unconscious. At times, also, the muscular twitchings may begin in the face. Sometimes the patient performs rotatory movements, or even walks or runs a few paces, before the convulsion develops.

A vaso-motor aura is also not infrequent; it may consist of flashes of heat or cold, or patches of pallor or redness appear in different parts of the skin. The aura may be limited to one limb or to the side of the face; the part affected becomes red, hot, and perspiring, or the reverse phenomena are noticed. This condition begins at the extremity of the limb and runs upward to the neck or head.

It is sometimes evident that epileptic children who are too young to describe their sensations, have an aura preceding their attacks. In several cases the mothers of such children have informed me that whenever the patient was about to have a fit, he would run to them, with a peculiar expression of dread, as if for protection. As far as I am able to learn, however, the aura in such young children is probably of a psychical nature, as I have been unable to obtain any history of objective changes in the little patients prior to the attack.

It not infrequently happens—in fact, we may observe in almost all chronic cases in which the fits occur with great frequency—that the aura *sometimes* makes its appearance, but is not followed by a convulsion.

At times this is merely an effect of the natural course of the disease; but, since the use of nitrite of amyl has come into vogue in the treatment of epilepsy, I have noticed more frequently than formerly that the inhalation of a few drops during the development of the aura has been instrumental in staving off the fit. I have also often observed that one of the first signs of improvement, under the use of the bromides, is the fact that the aura merely appears instead of the fully developed convulsion.

As we shall see at a later period, the aura sometimes furnishes valuable indications with regard to the origin of the epilepsy, and may thus prove very important with regard to treatment.

We shall now examine the phenomena directly connected with the convulsion more in detail.

The so-called epileptic cry is not observed in more than half the cases. In some instances it is so low that it cannot be heard at a distance of more than a few feet (this has been the rule in my experience), but at other times it is extremely loud. While I was interne in the Hospital for Epileptics and Paralytics, I was once awakened by a piercing shriek which appeared to emanate from the adjacent ward. Upon hurriedly entering the ward I found one of the patients in a convulsion; upon recovering, she told me that she had been unconscious of the utterance of this cry, although it had been loud enough to rouse me from a sound sleep at a distance of more than fifty feet from the patient's bed. In all the cases in which I was able to obtain any information upon the point, the patient was already unconscious at the time the cry was emitted. Dr. Reynolds,[1] however, observed one case which proves that it may be uttered while consciousness is still present. "The individual referred to was aware that he was making the noise, but he could not arrest it; he heard remarks made in another room, and made signals to his child to leave the room; but then lost all perception and volition."

It is probable that the cry is merely due to the vigorous tonic contraction of the muscles of expiration. The theory that it is the result of a sensation of terror or surprise has not been substantiated.

It appears from the united testimony of all observers that the face is usually pale at the onset of the attack, but there are numerous exceptions to this rule. Not infrequently the color of the face is unchanged until toward the close of the tonic stage, and, in a few exceptional cases, I have been informed by the friends of the patient that the face became of a bright red color at the beginning of the convulsion. If the clonic convulsions are at all marked, the face becomes dusky and puffed, and this condition is intensified the more vigorous the contractions of the muscles of the neck (trachelismus of Marshall Hall) have been. The pupils, as I have frequently had the opportunity to notice, dilate from the beginning and remain in this condition until the subsidence of the clonic stage. Some authors have observed that during this latter period they may alternately contract and expand, but they attain their normal size when the patient recovers consciousness. It has been said, as the result of ophthalmoscopic examination during the first period of the fit, that the fundus of the eye is in an anæmic condition. But, apart from the scarcity of these observations, the time for examination is so short, and the difficulties connected with its performance under such circumstances are so great that we are not disposed to lay much stress upon these results.

---

[1] Epilepsy; Its Symptoms, Treatment, and Relation to Other Chronic Convulsive Diseases.

The examination of the pulse during an epileptic convulsion is attended with great difficulties on account of the incessant movements of the muscles of the limbs. In some cases, indeed, the examination is rendered impossible.

At times we are unable to detect any appreciable change in the frequency or fulness of the pulse during the entire paroxysm. It appears, however, from the observation of numerous authors, that the pulse is usually small during the tonic stage, and in some instances it has been found absent at the wrist, although the carotids were beating visibly and forcibly. When the pulse can be felt during the clonic stage, it is rapid and full, and the heart at this time beats tumultuously.

Magnan [1] found, as the result of his investigations with the sphygmograph, that during the tonic period the arterial tension is increased, and the semitetanized heart beats with greater frequency. In the clonic period, on the contrary, the cardiac pulsations are accomplished with extreme slowness, and, at a later period, assume their normal rhythm, or may even become slightly accelerated. Voisin [2] states that the ascending branch of the sphygmographic tracing is higher than normal, and that marked dicrotism is manifested for some time after the cessation of the fit. He regards these appearances as sufficient to diagnose a case of simulation from a real attack of epilepsy. Magnan, however, believes that the sphygmograph will render no assistance in this respect.

These conflicting statements with regard to the condition of the pulse during and immediately after an epileptic paroxysm, show that no definite symptoms are manifested in this respect.

Let us now enter a little more closely into the character of the muscular contractions themselves. The first, or tonic stage, varies very much, both as regards extent and intensity. At times this period may be entirely absent, and the scene may be opened with clonic convulsions, which persist to the end of the seizure, or, on the contrary, this first stage may entirely predominate the scene, the clonic convulsions being either entirely wanting, or of a very mild character. The contractions may be present in every muscle of the body. Nothnagel even mentions a case in which the epileptic cry was followed by an active, whistling inspiration, such as is only seen in well-marked spasm of the glottis. It is sometimes noticed in the beginning of this stage that the face and neck are drawn to one side by tonic contractions of the muscles before the patient falls, after which the general convulsions make their appearance.

The clonic convulsions also vary greatly in their intensity. Sometimes they are so severe as to cause great bodily injury. Thus the tongue may be severely bitten, or even entirely divided, teeth may be broken, the lower maxillary bone or clavicle fractured, and various muscles may be ruptured from the intensity of their contraction. During this stage, the urine, semen, and fæces are often passed involuntarily. As we shall see in the chapter on diagnosis, the latter phenomena are sometimes very important in putting us on the track of nocturnal epilepsy.

Both stages of the convulsion, when severe, lead to great stasis in the vessels, and this may be followed by ruptures in the minute blood-vessels, giving rise to the development of a petechial eruption in the integument. This is usually visible around the outer angles of the eye, but, in severe cases, the entire face is involved and presents a speckled appearance.

---

[1] Gaz. méd. de Paris, 1877.

[2] Annales d'hygiène publique, April, 1868.

This may likewise be an important diagnostic sign in doubtful cases. It is a symptom which cannot be simulated, but unfortunately its absence does not exclude the diagnosis of epilepsy.

Unconsciousness is usually complete during both stages of the fit. The cornea may be touched without any response; the patients may fall into the fire and be horribly burned without their knowledge, and they have been known to drown in half an inch of water in cases in which they have fallen face foremost into a cistern or bath-tub. The history of a case which was very interesting in this respect was told me by my friend, Dr. Janeway. A man had been found dead and was brought to the dead-house without a previous history. Upon making the autopsy, Dr. Janeway found, as the sole cause of death, the presence of some dung in the larynx, which had given rise to asphyxia. This led him to suspect that the man had been an epileptic, and had probably fallen face foremost upon a dung-heap, during a convulsion. An investigation into the circumstances of the man's death proved the correctness of this surmise.

But consciousness may be entirely unaffected even during undoubted attacks of grand mal. In one case under my observation, the patient, a very intelligent man, assured me that during the first five years of his disease the seizures were accompanied with loss of consciousness, but that for a year afterward, although the body was strongly convulsed, he retained his consciousness throughout the attacks. I should, however, mention with regard to this patient that the epilepsy was undoubtedly due to a syphilitic cerebral gumma.

According to Romberg, reflex action may be retained throughout the attack, although consciousness is lost; this author states that pouring cold water on the body gives rise to reflex muscular contractions.

I desire to call attention to a peculiar anomaly with regard to the occurrence of coma, which is exemplified in the following case, whose parallel I have been unable to find in the literature of epilepsy.

Case I.—J. B., æt. 32 years, family history excellent, patient is married, and has seven healthy children; excessive sexual indulgence. He was perfectly healthy until about four years ago, at which time he was compelled, in the course of his work, to enter an ice-house for an hour and a half every afternoon, the patient always being in a state of perspiration at the time of entrance. About this time, while he was going to work one morning, he suddenly felt dizzy and weak, and a small amount of water-brash regurgitated from his stomach and ran out of his mouth. Since then he has had spells of this nature every day, or even two or three times a day. At times he had an aura immediately preceding the attack, consisting of a feeling of powerlessness in the left arm. At other times, he would experience a feeling of pruritus in the left eye, would then put his hand up to rub it and in a few seconds consciousness became partly clouded. In several of these attacks, the patient states that he thinks he lost consciousness entirely for a few seconds.

This variety of attacks lasted without being accompanied by other symptoms until January 1st, when, while sitting in a chair, the patient experienced a feeling of numbness in his left leg, and told one of his sons to rub the limb. This friction caused intense pain, but the patient was speechless and unable to tell his child to stop. Within a few seconds afterward he became unconscious, and tonic convulsions developed throughout the body. His wife states that these spasms were not followed by clonic convulsions. About three or four weeks afterward, the

patient had another fit while in his shop. For the first couple of months the attacks of grand mal came on about once a month, but they then appeared every two weeks. Until six months ago, the attacks of grand mal were preceded by an aura, consisting of a sensation of numbness in the left leg which rapidly rose to the arm. Within the last six months, the fit has been preceded by spasmodic separation of the maxillæ, so that the patient was unable to close his mouth, and by a queer sensation in the head. He always has sufficient time during the aura to lie down or seek a place of safety.

With the exception of the first two attacks of grand mal (occurring in January and February, 1879), the paroxysms have been of a remarkable nature. The convulsive phenomena corresponded exactly to the description of the grand mal which we have given in the beginning of this chapter, but the patient retained consciousness throughout the tonic and clonic convulsive stages. According to both his own and his wife's statements, he remembered everything which had transpired during the convulsions. As soon as the convulsions ceased, however, he became comatose for a period varying from five to fifteen minutes, the eyes being wide open during this period. While in this comatose condition, he can be partially roused but does not comprehend what is said to him. About three weeks ago (end of October), the patient had an attack in which he did not lose consciousness either during or after the convulsions. He is now complaining of intense headache from which he has suffered for the last two weeks; he had a similar attack in July, which lasted nearly a month. Under the use of bromide of potassium in thirty-grain doses, three times a day, the convulsions have ceased, but the headache from which he was suffering has increased in intensity, and at times the pain is atrocious. The patient's intellect has also begun to suffer ; memory is impaired, and at times he is almost childish in his behaviour. The appetite has become ravenous; the patient takes six or seven meals daily, and would eat more if permitted. The general physical condition of the patient is excellent; no deviations from the normal with regard to motion or sensation, are noticeable.

As we have previously stated, the duration of the coma following the convulsions varies from a very few minutes to upward of forty-eight hours. There does not appear to be any definite relation between the intensity of the convulsive phenomena and the duration of the subsequent coma. When the attack occurs during sleep, it is sometimes difficult to determine the difference between natural sleep and the epileptic coma. The patients usually lie quietly, though sometimes the breathing is noisy; if spoken to sharply, or irritated by a movement, they may be partially roused, speak or mutter incoherently, and then relapse into their previous somnolent condition. If left entirely to themselves, the coma continues longer than if they were disturbed from time to time.

As a rule, the patients return to their normal condition after they have recovered from the coma. Sometimes they feel much better mentally than they did for some time prior to the fit, as if the convulsion "had cleared the brain." In other cases they are dull, stupid, and irritable for a day or two, and, although they apparently act in a rational manner, may not retain perfect remembrance of the occurrences during this period, thus showing that consciousness is still slightly impaired. In more exceptional cases, the patients perform a series of automatic acts imme*diately* after the cessation of the coma. One of my patients, after she

recovers from the epileptic coma, begins to undo her clothes and goes about picking up various things. I observed another patient, under similar circumstances, rise from the floor and go to the door, the handle of which he grasped firmly in the hand and rattled violently to and fro. When I approached him he looked at me as if entirely unconscious of my presence, and allowed himself to be led to a seat. In a few minutes he had recovered consciousness and felt perfectly well, with the exception of a slight dull headache; he knew nothing of the occurrence of the fit until I informed him.

The stage of coma may also be immediately followed by a condition of mania, or this may not occur until the lapse of two or three days. We shall reserve the discussion of this feature until a later period.

The urine is usually passed in increased quantity after the occurrence of a convulsion, and at the same time presents a lighter color. It differs from the well-known hysterical urine, however, in the fact that the specific gravity is not diminished, whereas in the latter it becomes considerably lower, in fact I have seen it sink to 1,001.

Huppert[1] states as the result of numerous investigations that grand mal convulsions are invariably followed by albuminuria for a few hours.

Though albumen has been found in the urine under such circumstances by numerous observers, it does not by any means constitute an invariable rule. I have made several examinations with regard to this point and have always obtained negative results. We cannot therefore coincide in the view that a diagnosis of epilepsy may be excluded, if the urine passed after the suspected epileptic seizure does not contain albumen.

In some cases the patient has no sooner come out of one convulsion that another one begins, until finally they follow one another in such rapid succession that consciousness is not restored between the attacks. This constitutes the condition known as the *status epilepticus* (état de mal epileptique), and is of extremely serious import. We shall transcribe the history of this affection from Bourneville's article,[2] which is probably the best treatise on the subject extant. According to this author, the *status epilepticus* is characterized: 1st, by the frequent repetitions of the fits which may even become almost continuous with one another; 2d, by a variable degree of collapse, which may deepen into the most profound coma, unattended by any return of consciousness; 3d, by a more or less complete hemiplegia, developing after a variable duration of the symptoms; 4th, by increased frequency of the pulse and respiration; 5th, by a considerable elevation of the temperature, which persists in the brief intervals of the fits.

Bourneville divides the affection into two periods, viz.: a convulsive and a secondary meningitic stage.

The prodromata do not differ in any respect from those which are usually experienced by the patient prior to his ordinary attacks of convulsions. But the fits, instead of ceasing, follow one another rapidly, and before the epileptic coma has entirely disappeared another convulsion makes its appearance. The intervals between the fits become shorter and shorter until they run into one another, and finally the patient appears to be in one long-continued convulsion. The pulse is regular but usually small, the respirations become frequent and labored. The temperature begins to rise from the very beginning, and may rapidly reach a height of

---

[1] Virchow's Archiv. Bd. 59, Heft. 3 and 4.

[2] Études cliniques et thermométriques sur les maladies du système nerveux, 1873.

104, 105, or 107° F. and upward.[1] The skin feels hot and scorching, the face is covered with an abundant viscid sweat. Marked nystagmus is present, and the face and neck may be drawn to one side. The pupils are dilated either equally or unequally on the two sides, and do not react normally to light.

After the convulsions have lasted for some time, hemiplegia develops in a large number of cases, the face and limbs being affected as in ordinary cerebral hemiplegia. When the limbs on the paralyzed side are lifted up and then allowed to fall they drop like inert masses. The sensorial and intellectual functions are totally abolished, and the patients lie in a profound stupor which often deepens into coma.

Contracture sometimes occurs and involves either the muscles of the jaw, neck, or limbs. These contractures are especially produced after the cessation of the fits or when they are becoming more infrequent.

Death may occur in this stage in a condition of extreme cyanosis, due to the violence and frequent repetition of the convulsions. The number of the fits varies. In one of Bourneville's fatal cases the patient had twenty fits on the first day, forty-five on the second day, twenty-two on the third day, twenty-seven on the fourth day, and twelve on the fifth.

Recovery may also occur in the convulsive period, and in such cases, of course, the second stage is absent.

Secondary or meningitic stage.—At the close of the first stage, the convulsions become more infrequent, and then cease, but another series of symptoms develops. The intelligence is more or less affected, and the patient is in a state of hebetude or coma. At intervals this condition may be replaced for a few moments by maniacal delirium, which is often very violent, and is sometimes accompanied by hallucinations. The tongue is dry and coated, sordes is found upon the teeth, and nutrition is profoundly disturbed, the whole body becoming rapidly emaciated. At this time bedsores may make their appearance, varying from a simple erythematous patch to a more or less extensive necrosis of the skin.

The situation of these bedsores is not by any means so fixed as in cerebral hemorrhage or softening. They may involve: 1, the sacral region; 2, the buttocks; 3, the fold between the buttocks; 4, the skin covering the great trochanters. In addition to these symptoms, the temperature, which had began to fall after the subsidence of the convulsions, again rises. The entire group of symptoms may now increase in severity, the vital forces rapidly fail, and the disease progresses to a fatal termination. Or, on the other hand, the collapse diminishes, the functions of the skin become more normal, the tongue clears, the temperature again sinks, and in a few days the patient is restored to his ordinary condition of health.

Petit Mal.

This variety of epilepsy is much simpler in its manifestations than the grand or haut mal, and can be described in very few words. The usual course of the affection is shown by the following case:

Case II.—C. R., æt. 19 years; no hereditary influence discoverable. The patient had convulsions while teething. At the age of nine years

[1] In one of my own cases, the temperature did not rise above 101° F., though the patient had had at least twenty-five fits in rapid succession.

the patient began masturbating and has continued the practice ever since. According to her own admission she has masturbated once every night during this entire period, and the hesitating manner in which this statement was made, led me to believe that the habit was indulged in even more frequently. The epileptic convulsions began when the patient was fifteen years old; during the first year of the disease she had three attacks of grand mal, but none since that time. She has, however, had at least three or four attacks of petit mal daily since her sixteenth year. These seizures consisted of a simple loss of consciousness, attended with pallor of the face; the patient would sit and stare for a moment, in an unconscious condition, and then immediately recover herself. The attacks were frequently observed while the patient was sitting at table. While engaged in eating, her face would grow pale, the eyes assume a vacant stare, and the patient would become unconscious. If she had a knife or fork in her hand at the time, she would not drop it. After a few seconds, she would go on with her meals, not knowing that she had had a seizure. If an attack occurred while the patient was standing or walking, she would not fall or even totter.

This is the history of the mildest form of the attack, but very frequently the disease presents more marked symptoms. Thus, the duration of the attack may be longer; instead of lasting a few seconds, the fit may continue for a minute or two, and even from five to ten minutes, although the latter period is extremely rare. At times the attack is preceded by an aura, though this does not assume the importance that it does in the attacks of grand mal. The aura usually consists of vertigo lasting a few seconds, or of an epigastric pain. Sometimes it is preceded by an indescribable sensation in the head. One patient informs me that he experiences an indescribable, pleasant sensation immediately preceding the attack. The seizure may also be attended with slight convulsive movements in various parts of the body. In the mildest forms they are limited to the face and consist of a few grimaces, or of strabismus. The friends of several patients have informed me that the latter performed peculiar sucking movements with the lips and tongue during the attack. The following interesting case illustrates a form in which more complicated phenomena are produced:

Case III.—Charles A., æt. 21 years; family history unimportant. The patient had convulsions when he was three or four years old, and these continued off and on until the age of seven or eight years, after which they ceased. Since then the patient has been very healthy. During the past year, the patient has been employed off and on in a lunch-room at night and slept during the day; the room in which the patient worked was exceedingly hot. About ten weeks ago he first noticed spells coming on, attended with involuntary micturition. The attacks developed at night while the patient was at work, and occurred three or four times nightly. He was then thrown out of employment for a month, and during this time the spells came on during the day, the patient sleeping at night. The attack occurs in the following manner: while the patient is busily occupied, he suddenly, without any premonition, loses consciousness, his face reddens and subsequently grows pale, and he puts his hands to his head in an agitated manner; he then passes his water involuntarily. During the last week the patient has not micturated during an attack. Consciousness is restored in a few minutes.

There is no doubt that, in this instance, the excessive heat of the room was a prominent factor in the causation of the attacks. In accordance with my advice, the patient changed his occupation, and this measure alone produced a marked diminution in the number of the fits.

This case differs from the usual run in the fact that the face grew red in the beginning of the attack and then turned pale ; in the majority of cases, the face turns pale at the onset of the attack and then, if the paroxysm lasts for any length of time, grows dark or dusky.

Even more complicated movements than those just referred to are performed in these cases. One of my patients, if the attack occurs while he is seated, will rise from his chair and walk around as if looking for something. Cases have been reported in which the attacks occurred while the patients were performing on the piano or violin, and in which they continued to play in time, although entirely unconscious.

We may also include in this category the cases which are known as epileptic vertigo. These attacks are well shown in the history of the patient given on page 49. They consist merely of a feeling of vertigo, usually combined with faintness.

One feature which is frequently observed in this variety and which is extremely important with regard to diagnosis, is the fact that the attack is generally preceded by an aura of very short duration. This consists of a feeling of sinking in the epigastrium or of indescribable anguish. Sometimes the attack is accompanied by vague mutterings. One of my patients told me that he went off into a "dream-like" condition, which he could only explain by saying that everything around him appeared as if he were in a dream.

The paroxysms may alternate with well-marked attacks of petit mal or grand mal, and in such cases their nature soon becomes evident. In some, however, they form preliminary symptoms which precede the development of grand mal perhaps by several years, and in such instances it is extremely difficult to form a correct conception of the condition. But we shall discuss this subject more *in extenso* in the chapter on diagnosis.

Irregular Epilepsy.

This form of the disease is sometimes known as larvated epilepsy, but we think the term is misapplied; the paroxysms are as distinct as in either of the other varieties, the only difference being that they appear in a peculiar form. In the great majority of cases, the seizures are only observed in such patients that also suffer from grand or petit mal, and some individuals are exquisite examples of all the forms of the disease which we have described. In very rare instances the patient only experiences the irregular paroxysms, and one such case has come under my notice which I shall describe shortly.

Hughlings Jackson believes that these attacks are invariably preceded by a slight convulsion, but I cannot agree with him in this respect, basing my opinion on the following personal case :

Case IV.—A. B., æt. 28 years; married five years; family history entirely negative; the patient's general health has always been fair; menses regular but attended with a certain amount of dysmenorrhœa. The patient was accompanied by her husband, who desired to consult me

with regard to the advisibility of sending her to an insane asylum, as she was regarded as insane both by herself and husband. After making a thorough physical exploration and finding nothing abnormal in the thoracic or abdominal viscera (she complained of vague symptoms referable to these organs), I began to make an ophthalmoscopic examination. Just as I had brought the light of the mirror to fall upon the eye, and before I could catch a glimpse of the fundus, I noticed that the pupil dilated to the utmost, the face became pale, and the patient started back as if in affright. She then jumped up from her chair, looked with a terrified air at the window of my office and exclaimed, "Look at that black man. He has a dagger. He is going to kill me." She then walked a few paces, muttering some inarticulate words and in a few moments came to herself. The pulse was unaffected during the paroxysm. After the seizures the patient stated to me that she was absolutely unconscious of what had transpired. I then obtained a history of preceding attacks of a similar nature. Thus, on one occasion, the patient found herself in the immediate neighborhood of the East River, at a distance of several blocks from her house, whereas the last she remembered was that she had been at home. At another time she beat her husband, to whom she was devotedly attached, although she was entirely unconscious of what she was doing. Attacks of this kind had occurred for three years, and had come on without any exciting cause. Although I cross-questioned the patient and her husband very closely, I could not obtain the slightest evidence that she had ever suffered from petit or grand mal.

In this instance I had the patient under observation during the entire attack, and could not detect the slightest spasmodic symptoms. The series of phenomena manifested in this variety of epilepsy has been included by Hughlings Jackson under the apt title of mental automatism, and much more complex acts are performed than those reported in the history of the above-mentioned case.

Another very important peculiarity with regard to these seizures consists in the fact that a thought which was uppermost in the mind prior to the attack may exert some influence upon the actions performed during the unconscious condition. In addition, the patients may react, in a measure, to something which is said or done by those around them, and this apparently imparts a certain degree of volitional character to their acts, although they are, in reality, profoundly unconscious. This is exemplified in the following case:

Case V.—The patient in question was a young woman, who suffered from frequently repeated attacks of grand mal, and occasionally from irregular seizures. While sitting in my office, I saw her suddenly rise from her chair, walk forward a few paces, then turn around and attempt to walk into a closet. From the blank expression of her face I judged that she had lost consciousness. I walked up to her in order to examine her pulse and the condition of her pupils; she endeavored to push away my hand as I tried to grasp her wrist. I then ordered her, in a loud, peremptory tone of voice, to sit down, and my demand was immediately complied with. She remained seated for about a minute, muttering some indistinct words which I was unable to understand, after which she recovered. I inquired whether she was aware that she had had a fit, and she replied in the negative, stating that she knew nothing of what had transpired.

Hughlings Jackson[1] reports the two following cases, which are extremely interesting, with regard to this point:

CASE VI.—"I was sitting on his bed taking his history, he sitting by my side holding the inkstand. After asking him a question and getting no answer, I looked at him. He remained sitting, but his head was a little drooped, and his face slightly pale. He still kept hold of the inkstand, and after a moment moved as if to put it down. I tried to get hold of it, as it was tilting, but he pushed me away with the other hand. He was well again in about half a minute."

In the other case reported by Jackson, the history was furnished by the patient.

CASE VII.—"My wife and her sister being present, had been talking about supper, when it was agreed that my wife and I should have some cold fowl, and the sister some cocoa, if there were any fire. She went into the kitchen to see, and reported that there was one. Soon after I began to feel chilly after being so warm with gardening, and I said I would go down to the fire. I did so, and after standing there a few minutes, I felt symptoms of an attack and sat down, I believe, on a chair against the wall. And here my recollection failed, the next thing I was conscious of being the presence of my brother and mother (who had been sent for, as they lived opposite), and I have since been informed by my sister-in-law that she came into the kitchen, and found me standing by the table mixing *cocoa* in a dirty gallipot, half filled with bread and milk intended for the cat, and stirring the mixture with a mustard spoon, *which I must have gone to the cupboard to obtain.*"

"This caused them to send for my friends, to whom I talked, showing no surprise that they were there, and entirely unconscious of what I had been doing until told this morning."

These cases are of great importance from a medico-legal point of view. They prove that not alone may the series of acts which are committed during an irregular epileptic paroxysm appear to be logically connected together as they are in healthy individuals, but that they may also bear a certain relationship to desires expressed previous to the occurrence of the attack. In the last-mentioned case, the patient had agreed that his sister-in-law should have some cocoa, and during the paroxysm he was found stirring the cocoa, although he was entirely unconscious of so doing. But, as Hughlings Jackson remarks, if he had had a quarrel with his sister-in-law prior to the attack, we can very readily imagine that this fact may have acted upon him in his unconscious condition so as to lead him to commit murder. Kleptomania is not an infrequent symptom during these attacks, and has often brought the poor patients in contact with the arm of the law. It is very often extremely difficult to diagnose this variety, but we shall reserve the discussion of this part of the subject to the chapter on diagnosis.

We will finally refer to a number of conditions which have lately been placed in this category, and are known as epileptoid states.

Griesinger[2] has called attention to an entire group of cases in which

---

[1] West Riding Lunatic Asylum Med. Rep., vol. v., p. 105.
[2] Archiv. f. Psychiatrie u. Nervenkv. Bd. I.

the interparoxysmal symptoms are those usually regarded as characteristic of hypochondria or hysteria, but which differ from the latter in the fact that they are combined with vertiginous attacks. Griesinger regards these cases as examples of mild epileptic paroxysms, and strongly developed interparoxysmal symptoms. It is very difficult to know where to draw the line. Usually, when a patient suffers from the manifold symptoms of hypochondria, and complains at times of attacks of vertigo, the latter are attributed to cerebral congestion, to gastric disturbances, to constipation, etc. In the chapter on diagnosis we shall endeavor to point out the way in which a distinction may be made.

Another very curious phenomenon has recently been included in this group, viz.: paroxysmal attacks of sweating. H. Emminghaus[1] reports the following interesting case:

Case VIII.—The patient was a woman, forty-five years of age, whose courses were regular. While still a child she had suffered from well-marked epileptic convulsions, but these had disappeared spontaneously at the age of puberty. For a few years past, the patient has been attacked at times with paroxysms of sweating, which came on suddenly while she was engaged in some occupation and without any apparent cause. These attacks were associated with a feeling of weakness and slight giddiness, but after a few seconds all the symptoms disappeared, and the patient then felt entirely well.

In conclusion we will devote a few lines to what has been recently termed epileptoid "sleep-states" (schlafzustænde). In 1876, Westphal had called attention to a peculiar condition in which the patient sometimes suddenly lapses into a doze or even into a sound sleep, although he had been entirely wakeful immediately preceding the attack. Westphal left it undecided whether this should be regarded as an epileptoid state; but last year Franz Fischer, Jr.,[2] reported a case which he thought justified him in positively regarding this condition as truly epileptoid. In this case the patient would sometimes go off into a dose while standing or walking, and in a few of the attacks lost consciousness entirely. A case has also come under my own observation which appears to favor Fischer's interpretation of this phenomenon. In this case, which will be described more in detail in the chapter on etiology, the patient, who also suffered from undoubted grand mal, on one occasion, while sitting at table, sank into a dreamy sleep, which afterward lapsed into coma. Upon the restoration of consciousness, the patient found himself in bed, in which he had been placed by his family.

Interparoxysmal Condition.

In some cases the interparoxysmal condition in epilepsy is one of undisturbed bodily and mental health. I have seen some instances in which men of fine physical development, whose general health was perfectly normal, suffered from well-marked epilepsy, and I have sometimes thought that in these very patients the paroxysms were more severe than

---

[1] Ueber epileptoide Schweisse, Arch. f. Psych. 1873.
[2] Arch. f. Psych. 1878.

in others. Hercules is believed to have suffered from this disease, and hence arises one of its numerous appellations.

The mental condition may also be entirely normal. This statement is abundantly verified by the fact that such great men as Julius Cæsar, Mahomet, Napoleon, Newton, Petrarch, Peter the Great, and many others, were afflicted with the disease. But in the large majority of cases this does not hold good. In very many individuals the general health has already suffered before the disease makes its appearance, and in some the deterioration of health is, indeed, the exciting cause of the onset of the malady. Sooner or later, in the large proportion of cases, the health suffers to a greater or less extent, and in a few instances I have noticed that the deterioration in health promptly makes its appearance, even after a single fit. The patients usually complain of headache of a dull character and generally situated in the frontal region. The pain is not continuous, but does not remain absent for more than a few days at the utmost. In a few cases I have noticed that it acquired considerable intensity, and became almost continuous in character; it is then frequently situated in the occipital region. We may sometimes attribute the headache to the blows upon the skull which the patients receive during their frequent falls. The patients usually fall in one direction (very frequently on the face), and after a while, the periosteum becomes considerably thickened from the oft-repeated traumatism. Nervous tremors are noticeable in the muscles, especially in the arms and legs, and are sometimes very annoying. The tongue is coated and the patient complains of a bad taste in the mouth, and of oppression in the epigastrium after eating. As these unfortunates are, however, continually taking medicine, I am inclined to believe that the dyspeptic symptoms are due to the direct action of the drugs on the gastric mucous membrane. Bromide of potassium, for instance, which is now so generally employed, has a notoriously bad influence on the digestive functions. The bowels are usually confined, and this is often one of the chief complaints made by the patient. Some of them, in fact, stoutly affirm that the constipation exercises a powerful deleterious influence on the frequency of the fits. The appetite often varies a great deal, and is sometimes very capricious. While the patients at times eat sparingly, at others they have a voracious and ravenous appetite.

No careful investigations have been hitherto made with regard to the condition of the pulse and temperature, and my attention has not been specially drawn in this direction, but I have seen nothing which leads me to believe that there are any noteworthy disturbances in these respects.

The general appearance of confirmed epileptics is often characteristic. The former refinement of expression disappears, the cheeks become puffy, the lips look swollen and prominent, the eyes have a peculiar dull, staring look ; in fine, the lower nature of the individual becomes more apparent. Although this change of expression is not readily expressed in words, it is, nevertheless, very characteristic, and a large experience with this class of patients will often enable the physician to recognize an epileptic at the first glance.

The mental condition presents much more interesting phenomena. One of the first changes which is noticed in this respect is loss of memory. As so frequently happens in cerebral affections, the memory for recent events fails more quickly than that for remote occurrences. It has been the generally taught doctrine that the memory fails more rapidly in cases of petit mal than in grand mal, and until recently, I had implicitly *adopted* this doctrine on trust. But within the past year, my faith has

begun to waver in this regard as I have seen quite a number of striking exceptions to this rule. In one case in particular, the patient, a young woman of nineteen, has had from three to four attacks of petit mal daily for the last three years, and I found her memory entirely up to the average, while she herself informed me that it had never been better. I have seen quite a number of instances of this kind, though not so striking, within the last few months, but my attention has not been drawn to this point sufficiently long to confer any great value on these data.

The temper also undergoes a marked change. In a few cases the patients become very quiet and gentle, but, as a rule, they are the very reverse. This is especially noticeable in the wards of an epileptic hospital. It is extremely difficult to prevent the patients, whether male or female, from coming to open warfare. They are continually bickering and quarrelling on the slightest pretext, are extremely selfish and pilfer whatever they can lay their hands on. They are very anxious for sympathy (especially the female patients), and feign all sorts of complaints in order to draw the attention of the physician to themselves. Very frequently the preceding symptoms increase in intensity for a short time before the fit occurs. After the fits have lasted for a long time, and especially if they occur, as they often do, in series comprising from three or four to fifteen or twenty or more in a few days, the mental powers gradually deteriorate, the memory fails almost entirely, the judgment is lost, and the patient becomes entirely demented. In the latter cases, the mental decay is not gradual, but receives a considerable and sudden impetus after each series of fits. After they have sunk into this demented condition, the patients are hopelessly lost, so far as regards their mental powers.

The discussion of epileptic mental disorders is really within the province of the alienist, and I shall therefore content myself with giving a very brief summary of the remarks on this subject made by Krafft-Ebing,[1] the most recent writer of a systematic treatise on insanity. This author divides epileptic mental disorders into three classes :

1. Epileptic psychical degeneration.
2. Transitory psychical disturbances, usually attended with delirium.
3. Epileptic psychoses.

We have already described epileptic psychical degeneration above, so that it is unnecessary to refer to it again.

The *transitory attacks* of psychical disorder may last from a few hours to several days ; they occur either before or after the convulsions, especially after a series of attacks. They sometimes develop irrespective of any epileptic attack ; consciousness is either disordered or entirely lost. The nature of the acts performed varies greatly ; they are characterized by stupor and a sort of "somnambulistic" condition (daemmerzustaende).

The stupor is rarely the sole symptom ; it is usually combined with frightful delirium and hallucinations, sometimes with exalted religious ideas. At times, there are sudden outbreaks of violence, probably as the result of fear.

The "somnambulistic" conditions may occur before or after epileptic convulsions, or independently of them.

They include several varieties:

1. *Petit Mal.*—This is characterized by marked psychical depression, combined with terror and confusion of ideas. Under the influence of

[1] Lehrbuch der Psychiatrie. 1879.

these feelings, the patient wanders aimlessly around, believes that he is being pursued by his enemies and is destructive to himself or others. His consciousness of the acts performed while in this state is either partially or entirely lost; it is generally not connected with any previous convulsion and often develops out of the condition of psychical degeneration found in chronic epilepsy.

2. *Grand Mal.*—This is a furious mania, characterized by rapid development without any warning symptoms, by the extreme violence of the maniacal acts and by terrible delirium, in which fear plays the chief part. There are various gradations between this form and that described above as petit mal. The maniacal condition may alternate in these patients with periods of profound stupor. Grand mal usually occurs after convulsions, especially when they have appeared in a series. It may last from a few hours to several days, and after its cessation the patients retain no consciousness of the occurrences during the attack.

3. *Conditions attended with religious delirium.*—In this variety, the patient considers himself to be God, Jesus, an angel, etc., believes himself in heaven, receives communications from the Deity, delivers prophecies, etc. These ideas may change, and the patient thinks himself in danger of eternal damnation, sees the flames of hell, etc. In these cases, consciousness is only partly clouded, and the patient retains some knowledge of what has occurred during the attack.

4. *Conditions attended with dream-like ideas.*—In these cases, the patients appear to those surrounding them to be conscious of their acts, but, in reality, they are in a sort of somnambulistic condition. They act under the influence of various fanciful notions concerning themselves and those surrounding them. In a case mentioned by Legrand du Saulle, and which belongs in this category, the patient found himself on board of a vessel off Bombay, although he last remembered himself in Paris. All his actions in the interim had been a blank to him. In a case under my own observation, the patient left the city without any object in view, and when he recovered consciousness, found himself in the streets of Pittsburgh.

Epileptic psychoses.—Patients suffering from epilepsy may develop insanity, which differs in no respect from that observed in other classes of patients. But Samt has shown that there is also a specific form of epileptic insanity, and this author states, "that every patient who is in a condition of stupor, kneels in terror before the physician, calls him 'God,' and at times becomes destructive," may be regarded as suffering from epileptic insanity. Samt divides the disease into several varieties, but the nature of this article will prevent us from entering any further into a consideration of the subject.

CHAPTER II.

ETIOLOGY.

EPILEPSY is one of the most frequent of all nervous diseases. Reynolds states that it constituted seven per cent. of all the cases of nervous disease which he met with in hospital practice. This agrees very closely with the results of my own observation. Among 2,299 patients who attended the clinic for nervous diseases in the Bellevue Out-Door Department during the last three years, 156, or a little over six and three-fourths per cent. suffered from the various forms of this disease.

The causes of epilepsy may be divided into two classes, viz.: the predisposing and exciting.

PREDISPOSING CAUSES.

Heredity undoubtedly constitutes the most important factor in this class, though its frequency is variously estimated by different authorities. Delasiauve [1] found that in 300 cases, no evidence on this point could be obtained in 167, and that among the remaining 133 patients there was a neuropathic tendency in only thirteen, or a little less than ten per cent. In thirty-eight cases under Reynolds's [2] care, twelve presented a history of nervous disease in the family, or a proportion of thirty-one per cent. In 306 of Echeverria's cases, eighty had a neuropathic family history. I have notes on this point in one hundred and twenty-four of my own cases, and find heredity as an etiological factor in thirty of these patients, or in twenty-three per cent.

Gowers,[3] who has had an extremely large experience at the English National Hospital for the Paralyzed and Epileptics, found that among 1,250 epileptics, in whom this point was carefully investigated, an hereditary neurotic tendency was present in 452, or thirty-six per cent.; among these, the female sex numbered fifty-seven per cent., and the male, forty-three per cent. This author has included in his statistics only such cases in which there was no reason to suspect cerebral tumor, chronic meningitis, and syphilitic or other organic disease.

Martin [4] states that almost all the children of epileptics die during the first years of life. This is one reason why the disease is not transmitted more frequently, as it is to be presumed that a considerable proportion of these children would be similarly affected if they had lived until a more advanced age.

The powerful influence of heredity is also shown by the fact that

---

[1] Traité de l'epilepsie.
[2] Epilepsy; its Symptoms, Treatment, etc., 1861.
[3] British Med. Journal, March 6, 1880, et seq.
[4] Annales medico-psychologiques, T. xx., p. 364.

guinea-pigs, which have been rendered epileptic by experimental means, may transmit the disease to their offspring.

Finally, in estimating the importance of this factor, we must take into consideration the fact that the relatives of the patient, and the patient himself, often endeavor to conceal the history of any antecedent nervous disorder in the family, and frequently attempt, by hook and by crook, to adduce some palpable physical factor as the cause of the disease. In interrogating hospital patients we must also bear in mind that stupidity often reigns supreme, and that a strict cross-examination may reveal the presence of disease which has been previously denied.

We do not imply by the term hereditary influence, that the ancestors must have had epilepsy. In fact, any nervous disease in the parent may produce epilepsy in the children.

Thus, the existence of hysteria, chorea, insanity, obstinate neuralgia, or even simple nervousness in the parent may be transmuted into epilepsy in one of the descendants. It is true, however, that the nervous disease exhibited in the parent is often transmitted directly to the offspring. Thus there are numerous cases on record in which a tendency to commit suicide has been transmitted from parent to offspring (usually from father to son) for several generations, and, strange to say, this tendency is frequently manifested at about the same period of life in all the individuals of the family. But in many instances, the neuropathic tendency will give rise to the production of epilepsy in one child, chorea in another, and idiocy in a third. This transmutation of disease is also often shown in the same individual, so that a patient who was choreic in childhood, becomes epileptic in manhood, and insane in later life.

In addition to these diseases, alcoholism in the parents may exercise a similar baneful influence on the offspring. Quite a number of cases are recorded in which the birth of an epileptic child was due to the occurrence of conception while the father was intoxicated. That this is not a mere coincidence has been shown by the fact that the children who were born later, and in whom conception did not occur under such circumstances, remained perfectly healthy. In two of my cases, also, I discovered that the father was the victim of a periodical and uncontrollable desire to drink, and that after such a debauch had ceased, he would remain entirely abstinent until again seized by the uncontrollable craving. There is no doubt that inebriety may be transmitted, and I have myself seen a few cases in which several examples were presented in the same family. I also see no reason for doubting that it may act directly in the production of epilepsy in the offspring. The hereditary influence sometimes skips one, and perhaps even two generations, and then reappears in the second or third. In one case under my observation, the son escaped, while of the latter's seven children, all suffered from epilepsy. In another of my cases, the only hereditary history obtainable was that of insanity in the great-grandaunt; this reappeared in two of the great-grandnephews as chorea and epilepsy respectively.

Some of the older authors believed that phthisis in the parents is capable of developing epilepsy in the progeny, and this view has been more recently supported by Echeverria. The notion has probably arisen from the great frequency of phthisis; at all events, we would be unwilling to accept this statement unless substantiated by careful statistics.

It would appear that in those individuals in whom the epilepsy is due to hereditary influence, the disease makes its appearance, in a large number *of cases,* before the fifteenth year of life.

Reynolds found that among twelve patients in whom an hereditary taint could be traced, in ten the disease appeared before the age of fifteen years, and in two between the ages of fifteen and twenty.

My own experience somewhat corroborates that of Reynolds. In twenty-six cases with a neuropathic family history, the disease developed nineteen times before the fifteenth year, five times from the fifteenth to the twenty-sixth year. In one case the disease began at the age of sixty-seven years, in a vigorous man, enjoying excellent health. In this patient, the hereditary taint was shown by the development of insanity in two cousins.

Gowers has drawn different deductions from his statistics, and has come to the conclusion that heredity possesses very little less influence in the production of epilepsy during adult life than it does in childhood. This is so contrary, however, to the general opinion of the great mass of observers, that we are unwilling to accept this opinion unless substantiated by the experience of others.

From a careful analysis of his cases, Reynolds concludes "that hereditary taint is not without influence upon the character of the attacks; but that the influence it exerts is favorable to the development of epilepsia gravior, rather than epilepsia mitior."

I will conclude my remarks on heredity by giving the family history of an interesting case under my observation. The father was a periodical drinker; by his first wife (who was healthy) he had four children, two daughters, one of whom was insane, and another imbecile (neither of these had children); and two healthy sons, one of whom had an epileptic, and the other an insane child. By the second wife (also healthy) he had three sons, one of whom died of epilepsy, and another was epileptic, and had an insane child; the third son was healthy and had seven children. All of these had fits in infancy; one was a confirmed epileptic, and another suffered from epileptic insanity.

Sex.—The prevailing opinion, especially among the older writers, has been that the disease is more common among females than among males. Several of the more recent authors, however, are opposed to this view. Among eighty-eight cases, Reynolds found forty-nine males and thirty-nine females. Eulenburg[1] found seventy-three in males, and fifty-nine in females; Nothnagel states that it occurs in about equal frequency in both sexes. I have observed seventy-five cases in males and fifty-nine in females. Gowers, whose statistics include 1,450 cases, found that there were 114 females to 100 males; after the age of thirty years, however, the proportion of males is greater than that of females, and this disparity increases after forty. It is evident, therefore, that sex possesses very little, if any, influence on the development of the disease.

Age.—This factor is more important as an etiological element than the preceding one. In Reynolds's experience, the disease appeared in forty-eight patients during the first seventeen years of life, in fourteen between the ages of seventeen to twenty years, and in fourteen from the ages of twenty-one to seventy years. Among Gowers's patients, 12½ per cent. began during the first three years of life, 29 per cent. in the first ten years, 46 per cent. between the ages of ten and twenty years, and 15.7 per cent. between the ages of twenty and thirty years. In my own cases, I found that among ninety-three patients, the disease appeared in the first seventeen years of life in fifty-seven patients, from seventeen to

[1] Lehrbuch der Nervenkrankheiten. 1878.

thirty years in sixteen, and from thirty to sixty-seven years in twenty individuals. It has been supposed that the later the disease makes its appearance the more is it due to causes other than heredity, although, as we have seen, Gowers holds to the opposite opinion. In the twenty patients in whom it first appeared between the ages of thirty to sixty-seven years, in nine no cause could be obtained, in two it was due to traumatism, in two to syphilis, in two to alcoholism, in one to sexual excess, and in one each to exposure, fright, heredity, smoking. This exhibit runs counter to the current opinion according to which the development of epilepsy after the thirtieth year of life is usually an indication of cerebral syphilis.

I must, however, mention in this connection that I have omitted from the category of syphilitic epilepsy, all those cases in which the patients presented other well-marked tumor symptoms, such as paralysis of the ocular muscles, facial paralysis, atrophy of the optic nerves, etc.

It is well also to be on our guard when confronted with epilepsy which has developed late in life, lest the epilepsy is only one symptom of some organic cerebral affection, and the older the patient is at the onset of the disease, the more careful must be our search for other symptoms. In two of my cases, however, the disease first appeared at the ages of fifty-seven and sixty-seven years respectively, and their course showed that they were evidently idiopathic epilepsy. In the former case the patient, who was in tolerable health and presented an excellent family history, worried a great deal about the intended removal of her daughter from the city, and on the very day of her departure, the patient had the first attack of grand mal. The attacks then occurred at irregular intervals during the next two years until she came under my care. Under the use of bromide of potassium, the convulsions entirely ceased and have not returned for the past year.[1]

The second patient, a healthy farmer, in whom the only etiological factor obtainable was insanity in two cousins (showing the presence of an hereditary taint), had the first attack at the age of sixty-seven, after which they appeared at short intervals for three years. During this period he had upward of 150 attacks, all of them being well-marked paroxysms of grand mal. For nearly a year the patient suffered from epileptic mania, and was very violent, excitable, and destructive. The last attack occurred during the summer of 1877, and since that time the patient has been in excellent health, with the exception of an attack of bronchitis, and his previous mental condition has been entirely restored.

Reynolds mentions a case of pure epilepsy which developed at the age of seventy-one, and Heberden another at seventy-five. Other cases have been reported at such an advanced age, but they form decided exceptions.

We shall now direct our attention to the accidental or exciting causes, which are much more numerous than the predisposing.

Alcoholism.—This is, on the whole, a very infrequent cause of the disease, and was observed in few of my cases. Among 608 cases analyzed by Gowers with reference to etiology, only thirteen were traceable to chronic alcoholism. Alcoholic epilepsy may develop either in individuals who are in a continual state of semi-intoxication or in those who go on occasional sprees, which terminate in mild delirium tremens.

[1] As this book is going through the press, the patient died with symptoms which I referred to a fatty and dilated heart. Unfortunately I was unable to obtain an *autopsy*.

We must, however, exercise caution in pronouncing drink an etiological factor. In one instance a patient was referred to me by a medical friend as a case of alcoholic epilepsy, but, upon careful questioning, I found that although, at the present time, the fits only appeared when the patient went on a spree, the attacks had occurred during childhood, and that there was an hereditary taint in the family. Cases of this kind are not unfrequently observed, and I have several times noticed that in patients in whom the disease had been latent for several years, the fits again recurred with the former frequency, after the patient had been drinking heavily for some time. In the majority of my cases, the attacks which were brought on by drunkenness occurred during the night, and were always of the nature of grand mal. Magnan has shown that absinthe possesses a great influence on the production of epilepsy, but Legrand du Saulle believes that the nature of the stimulant is immaterial, that brandy, whiskey, and wine are alike potent in this regard; this author regards the alcohol as the only efficient factor. I am unable to offer any opinion upon this question, as the only cases which have come under my notice, in which the convulsions could be attributed to intoxication, were due to drinking whiskey, brandy, or beer. Fortunately, the habit of drinking absinthe has not found much favor in this country, and we will therefore be unable to institute any comparisons between its effects and those of the stimulants habitually drunk here.

Sexual excesses.—In one of my cases, sexual excess constituted the only traceable cause. The patient, a middle-aged man, had had intercourse six or seven times a week since early manhood, had a perfect family history, and was entirely well in all other respects. The older authors laid great stress upon this factor as well as upon sexual continence and masturbation, as active causes of epilepsy. The activity of these causes has been greatly exaggerated, especially with reference to masturbation. A very intelligent and observant patient in the male pavilion of the Epileptic and Paralytic Hospital, told me that all the patients in the ward practised masturbation, himself included, but that he had only begun the habit after he was attacked with epilepsy. The reason that the practice is so widespread in an epileptic hospital is obvious. The disease itself produces a depression of the moral stamina of the patients, and the character of the affection prevents, in great part, the exercise of the sexual functions in the natural manner. My own observation in the female pavilion of the hospital has also taught me that masturbation is very extensively practised by female epileptics. But these facts are far from indicating that the masturbation was the cause of the epileptic attacks. Although I have carefully investigated this phase of the question for several years past at my clinic in the Bellevue Out-Door Department (in which the facilities for such investigations are, of course, much greater than in private practice), I have been unable to find a single case of epilepsy which I could with justice attribute to this cause. When tempted to believe that a case of epilepsy is due to this habit, we should bear in mind how common the practice is among the young of both sexes, and that, if the solitary vice possessed any marked degree of influence on the development of epilepsy, the latter disease should be much more frequent than it really is. I remember one patient, a maiden lady, thirty-one years old, who confessed to me that, since the age of nine years, she had masturbated at least once every night. The only effect produced on this patient was the development of symptoms of nervous exhaustion, or, as it is now fashionably termed, cerebral neurasthenia.

In a few instances the first epileptic convulsion has occurred during the performance of the sexual act and only appears when sexual intercourse is attempted. This occurs both in males and females, and is sometimes observed when the genital organs appear to be entirely normal. In rare cases this is due in the female to the irritation of hypersensitive portions of the genital tract, but these cases really belong to the category of reflex epilepsy, and will be discussed under that heading. As a rule, the convulsions which at first only occur during the sexual act, afterward appear apart from this exciting cause, and the disease then runs the course of ordinary epilepsy. But, even in cases of this character, we should always carefully examine the patients with regard to the presence of some hereditary predisposition.

A few cases have been reported by trustworthy observers in which the epileptic attacks appeared to be due to the irritability produced by absolute continence, and in which marriage and regular sexual intercourse caused the disappearance of the disease. But these examples are very exceptional and they should not lead us to advise matrimony in young unmarried epileptics of either sex. We must remember that even though marriage prove remedial in the parent, the disease or some other neurosis may be transmitted to the offspring of such a union. At all events the patient should be informed of the possibility of such an occurrence, and the responsibility of the decision thrown upon his own shoulders. It is unnecessary to state, however, that patients will rarely be deterred from marriage by the possibility of a contingency of this nature.

Reflex epilepsy.—In ten of my cases the disease was due to reflex causes, which consisted: in six cases of injuries to the head; in three, to various other parts of the body (leg, abdomen, back); and in one, to pregnancy. In one instance the causal relation between the injury and the development of the epilepsy was very evident. The patient in question was a young man, nineteen years of age, who presented no ascertainable hereditary taint, and had shown no evidences of nervous disease until the age of two years, when he fell from a carriage, striking upon his head. He was rendered unconscious by the blow for upward of an hour and a half, and the same night was attacked by an epileptic convulsion. These attacks recurred at intervals until the age of seven years, at which period they ceased entirely and remained absent until the age of seventeen (two years ago) ; during this time the patient enjoyed excellent health. He then had another fall upon the head, after which the epileptic attacks reappeared and have continued up to the present time. As a rule, epilepsy will not develop from a blow upon the head, unless the injury has been sufficiently severe to produce unconsciousness for a considerable period.

In one instance, in which I succeeded in obtaining an autopsy, the affection was caused by the pressure of exostoses upon the brain. These growths, which measured about an inch in length at their base and three-fourths of an inch in thickness, projected from the frontal bone and were symmetrically situated on each side of the falx cerebri ; they had produced corresponding depressions in the anterior portions of the superior and middle frontal convolutions. The only nervous symptoms existing during life consisted of the epileptic attacks; the patient died of pericarditis and fatty degeneration of the heart.

Similar results may follow from depression of the skull, and from irritation of the dura mater or surface of the brain by a splinter of bone. Epilepsy is also not uncommonly observed in pachymeningitis, cerebral

gummata, and other tumors of the brain, especially when they are situated on the anterior portions of the convexity. But in these cases, the epilepsy is usually combined with other cerebral symptoms, to which we shall refer in the chapter on diagnosis. In one patient, the disease began a year and a half after an attack of cerebral hemorrhage, but the previous history was so indefinite that I am unable to state positively whether the latter affection acted as a cause of the epilepsy.

Injuries of the peripheral nerves also act as etiological factors. This is usually due to lesions of the sensory and mixed nerves, but cases have also been reported in which the epilepsy was secondary to an affection of purely motor nerves. In these cases neuritis almost always develops in the injured nerves, and neuromata are sometimes observed upon them.

Thus, in one case the disease followed the development of a neuroma in an amputation stump. In a case which came under my observation, the epilepsy developed after an injury to the outer aspect of the left leg; no symptoms of neuritis were manifested at any time. The epileptic attacks were always preceded by a motor aura which began in the injured leg and rapidly spread up the left side of the body. Although there was no pain or tenderness over the injured part, the convulsions rapidly became more infrequent from the local application of the constant galvanic current three times a week. I lost sight of the patient, however, within a couple of months after beginning the treatment, and I am therefore unable to report upon the final termination.

These cases form the analogues of the epilepsy produced in various animals, especially guinea-pigs, by certain experimental procedures, such as blows on the head, section, or other injuries of the peripheral nerves, etc., and which we shall describe at a later period in the chapter on pathology. As in animals, the cases in man which are due to traumatism present, in extremely rare cases, an epileptogenic zone, *i.e.*, a region whose irritation will give rise to the production of an epileptic convulsion. My own cases did not present this symptom, and I shall therefore give the following abstract of a very interesting observation of this nature reported by Dr. Neftel.[1]

Case IX.—H. W. K., æt. 24 years, entirely free from any neuropathic tendency; was previously in perfect health. In July, 1869, he was struck on the head, during a riot, with a loaded cane. He fell senseless to the ground and, while in this condition, received several more blows upon the skull. The first blow struck the right side of the forehead over the frontal eminence, but no external marks of contusion were present. The patient remained unconscious for seventy-two hours. During the second week after the injury he remained very feeble, and, after the slightest exertion, lost consciousness, which remained suspended for ten minutes and was attended with epileptic convulsions. After the lapse of three months the patient began to walk about, but had several attacks while in the street.

He began to suffer from headache as soon as consciousness had been restored after the accident, and this has continued uninterruptedly ever since. The pain is situated on the right side of the forehead and in the right eye, the most sensitive part being the right external frontal crest. *If considerable pressure is made over the sensitive zone, the pains become intensified to an intolerable degree, and the patient falls to the ground in*

[1] Arch. für Psychiatrie. VII. 1877, p. 124.

an unconscious condition; convulsions then make their appearance which were regarded, by the physicians who saw them, as epileptic in character. At rare intervals, attacks of unconsciousness develop, although no pressure has been made upon the epileptogenic zone. During the spontaneous pains, the skin of the painful region is analgesic.

This patient was treated by local applications of galvanism, which soon produced a remarkable improvement, but Dr. Neftel lost sight of him after the current had been applied thirty-one times.

Dr. Neftel believes himself "justified in concluding, or at least in surmising, that those cases of epilepsy, in which an epileptogenic zone is present, have been caused by traumatism." The case at least teaches us, apart from its intrinsic interest, to exercise great care in an examination of patients suffering from traumatic epilepsy in searching for the presence of an epileptogenic zone. This remark is especially applicable with regard to the epilepsy of young children, who so frequently suffer from blows on the head.

In a certain number of cases epilepsy is due to teething. Gowers found that among 180 cases beginning during the first three years of life, seventy-two developed during dentition. We not infrequently notice that epileptics have suffered from eclamptic attacks during the period of dentition, and some authors believe that frequently recurring eclamptic convulsions may produce an "epileptic habit" in the brain and thus give rise to the independent existence of epilepsy. But the eclampsia of childhood is so overwhelmingly more frequent than epilepsy that we are very skeptical with regard to its efficiency as a cause of the latter affection.

Diseases of almost all the organs of the body may act as exciting causes of epilepsy. Several cases are reported in which various affections of the ear acted in this manner. Fabrice de Hilden reported a case in which epilepsy was due to the presence of a glass bead in the ear and was cured by its removal. Schwig [1] reports the following interesting example:

Case X.—The patient was eleven years old; one year and a half previously he fell from a baby carriage, struck upon the right cheek and ear, and was dragged along for a short distance on a gravel road. During the next few days the patient complained of slight stitches in the right ear, and five or six weeks later, an epileptic convulsion suddenly developed without any warning. The child had been very quiet and subdued since the accident, in contrast to his former lively spirit. The epileptic attacks were repeated at varying intervals. Upon examining the ear, the auditory canal was found entirely occluded by a hard substance. The mass was removed by a pair of forceps, and during this operation the patient was seized with a convulsion; in the centre of the mass was found a small stone with sharp angles. A period of ten months has elapsed since the removal of the foreign body, during which no convulsion occurred. The boy, who was previously pale and had a stupid expression of countenance, is now the picture of health.

Moos [2] has entered fully into the literature of this subject, and from his article it appears that, apart from foreign bodies in the ear, epilepsy may also be produced by inflammatory affections of the middle ear, and caries of the bones of the ear.

---

[1] Archiv f. Ohrenheilkunde. Bd. XIV. 1878.
[2] Arch. f. Augen u. Ohrenheilk. IV. 2.

Affections of the respiratory passages may also give rise to epilepsy. Sommerbrodt [1] reports a case in which epilepsy was caused by the presence of a fibroma on the left vocal cord, and in which the attacks ceased after the extirpation of the tumor. Charpignon [2] observed a case in which a foreign body in the bronchi produced epilepsy. Charcot observed epileptic attacks in an old man suffering from chronic bronchitis and emphysema, in whom the attacks were always preceded by a dry cough and a feeling of titillation in the neck below the larynx.

Epilepsy also appears to be connected, in some instances, with disorders of the female genital apparatus. Numerous gynæcologists believe that amenorrhœa is a not infrequent cause of epilepsy. Graily Hewitt [3] mentions an interesting case of this kind, in which the fits ceased upon the appearance of the first menstrual epoch. I have, however, seen so many cases in which epilepsy developing during childhood was combined, at the age of puberty, with amenorrhœa, and in which, at a still later period, the menses appeared and continued regularly without any apparent effect upon the frequency of occurrence of the epileptic convulsions, that I have become somewhat skeptical as to the existence of any causal relation between the two affections. The period of menstruation frequently has considerable influence on the occurrence of the convulsions. Sometimes they develop immediately prior to the establishment of the menstrual discharge, at others they do not appear until the latter has lasted for a few days. I have found, however, as a general rule, that even when the epileptic attacks were at first connected with the menses, they occurred independently of them after the disease had lasted for a certain length of time.

In one case the convulsions appeared to be due to anteversion of the uterus, as they disappeared after the organ was restored to its normal position. In the following case, the epilepsy seemed to be the result of the reflex irritation caused by the presence of a fœtus in utero.

Case XI.—Elizabeth W., æt. 18 years, single; no hereditary influence. When six years old the patient had scarlatina, and since then has been perfectly well until the beginning of this year (this history was taken September 5, 1876). She began to menstruate at the age of fourteen years, and the courses have always been regular. On February 1, 1876, the patient was attacked with diphtheria in a very severe form, which was attended with regurgitation of fluids through the nostrils. She had recovered by the 1st of April. After recovery, the patient took a walk one afternoon in the month of May, and states that she then caught cold. The same afternoon she had the first epileptic attack (grand mal), followed by another one during the night. On the next day there was a recurrence of the diphtheria, and during the entire period of this relapse she had quite a number of convulsions. Since then she has had them almost every night, and often also in the day-time, with the exception of three and a half months, from the middle of May until September 1st, during which time she was in the country and only had two fits. Since September 1st she has had them every day and night—sometimes as many as two in the afternoon (never in the morning) and three or four at night.

Upon physical examination the patient was found to be in the fourth month of pregnancy, and upon comparing the dates of the cessation of

---

[1] Berl. Klin. Wochenschr. 1876.
[2] Gaz. des hôpitaux, 1876.
[3] Diseases of Women, 1874, p. 427.

the menses and the appearance of the first fit, it was found that the latter had developed within a couple of weeks after impregnation had occurred. At the time of the first convulsion the patient was unaware of her condition, so that the attack could not have been due to worry on this account. Since her knowledge of this fact, however, she has been exceedingly worried, and this has undoubtedly had considerable influence on the frequency of the fits. I kept the patient under observation until she had reached the middle of the seventh month of pregnancy; during this entire time the urine had been carefully examined on several occasions with entirely negative results. During the last month the patient's memory became worse and her general mental condition failed somewhat. I then consulted two eminent gynæcologists as to the propriety of bringing on premature labor, which I believed justifiable. My consultants, however, entertained a different opinion, and my suggestion was not carried out. Soon after I lost sight of the patient.

It may be claimed that, in this case, the disease was due to the diphtheria, but I doubt this, for the following reasons: In the first place, the patient had entirely recovered from the diphtheria at the time of the first convulsion; secondly, the convulsions began within a couple of weeks after impregnation; and finally, they increased in frequency toward the end of pregnancy. The latter fact was not due to the increased worry, as latterly the patient was becoming reconciled to her lot, her condition was unsuspected by her family, and she had made arrangements to be confined in a neighboring city, in order to obviate risk of detection.

Rosenthal[1] mentions a striking case of reflex epilepsy, relieved by local treatment.

Case XII.—"A young woman, twenty-four years of age, who had been previously healthy, suffered, at the end of the fourth month of marriage, after the performance of the sexual act, from acute pains in the abdomen, which were soon attended by convulsions, combined with loss of consciousness. During the following weeks the patient abstained from coitus and enjoyed perfect health. When she again indulged in sexual intercourse the epileptic seizures returned and soon began to occur spontaneously, at first only at the menstrual epochs, and, at a later period, irrespective of the menses. The patient did not place herself under medical care until after separation from her husband. Upon examination a very sensitive point was found at the anterior and inferior portion of the vestibule of the vagina, at the level of the remains of the hymen and of the adjacent mucous membrane. An attack of epilepsy could be invariably produced by pressing upon this point and even by touching it lightly with nitrate of silver; if the examination were prolonged the attack lasted much longer. The uterus was normal and insensible to pressure; there were no symptoms of hysteria. Ferruginous mineral waters and local treatment proved ineffectual, but the attacks of epilepsy disappeared after the excision of the sensitive parts, and have remained absent for the past two years."

Disturbances of the digestive organs may also act as causes of the outbreak of the disease; thus, it has appeared after an attack of subacute gastritis, overloaded stomach, and from the presence of worms. But the

[1] *Diseases* of the Nervous System, 1879, p. 340.

influence of gastro-intestinal disorders upon epilepsy is less evident and frequent than that of any other series of reflex causes.

Fright, mental excitement or anxiety, may also act as etiological factors. Among 608 cases Gowers found 157 which were ascribed to these causes. These cases were more numerous among females than among males, and this is but natural, as the former are much more emotional than the latter. A small number of examples have been reported in which the excitement attendant upon seeing an individual during a convulsion has been sufficient to give rise to an attack. In some instances, cases which had developed in this manner recurred spontaneously afterward.

The last series of causes of epilepsy is composed of those factors which affect the general system. Foremost among these are the infectious diseases, although but little mention is made of them in the various works on this affection, except by Gowers, who reports nineteen cases due to scarlatina. Among my own cases the infectious diseases immediately preceded the attacks in four cases, one of which occurred immediately after scarlatina, another during the course of scarlatinous nephritis, a third during convalescence from typhoid fever, and the fourth after diphtheria. In the second case referred to, in which the first attacks occurred during scarlatinous nephritis, the paroxysms might perhaps have been regarded as uræmic in their nature, were it not for the fact that they were attacks of petit mal, and occurred with extreme frequency (every day, and sometimes four or five times a day). After the disease had continued for three years attacks of grand mal occurred.

We sometimes, on the contrary, find that an intercurrent infectious disease, occurring during the course of epilepsy, favorably modifies the progress of the latter. Thus, in a female patient in whom the convulsions had occurred with great frequency during childhood, the disease disappeared for a period of ten years, after passing through an attack of typhoid fever, and only reappeared after the birth of her second child, while she was worrying greatly over the bad habits of her husband.

Syphilis sometimes acts as a cause of epilepsy, the convulsions usually developing during the tertiary stage. In only two cases under my observation did the attacks occur without being complicated by other cerebral symptoms, and in these I am therefore inclined to believe that the epilepsy was due to the direct action of the syphilitic virus upon the brain. In the vast majority of cases, however, the convulsions of the tertiary stage only constitute one of the symptoms of cerebral syphilis, and we shall again refer to them in the chapter on diagnosis.

Until very recently it was supposed that syphilitic epilepsy only occurred during the tertiary period. Fournier[1] has, however, collected twelve cases (chiefly in females) in which the convulsions began during the first months of secondary syphilis, and were accompanied by secondary manifestations (roseola, mucous patches, etc.). The epilepsy was relieved in all cases under mercurial treatment. The following case will serve as an illustration:

"The patient, a young, well-nourished woman, contracted syphilis three months previously. Upon admission to the hospital she was found to be suffering from enlargement of the inguinal glands, and had the remains of the primary induration on the labia majora; a papulo-erosive syphilide of the vulva and general roseola were present. A few days

[1] Annales de dermatologie et de syphiligraphie, 1880, pp. 16—24.

after her entrance into the hospital she was suddenly seized with two convulsions, occurring in rapid succession, after which she remained in a semi-comatose condition until the next morning. These two attacks were seen by the interne, who stated that they were typical epileptic convulsions. Upon the following day the patient had another attack, which was witnessed by Fournier. There was no hereditary neuropathic tendency or any other discoverable cause of epilepsy. The patient was put upon the protoiodide of mercury and rapidly improved; she was seen a year later, but there had been no return of the convulsions."

It has also been found in a few cases that epilepsy may result, in children, from congenital syphilis.

Overexertion, whether mental or physical, is also regarded as a cause. I have seen two examples of each variety. In one of the latter cases the first convulsion appeared after a day's tramp of forty miles on the Western prairies; in the other, the first attack came on after the patient, a girl of sixteen, had run a considerable distance, and was tired and overheated in consequence.

In one case the disease was distinctly traceable to excessive smoking. The patient was a man, æt. 45 years, in whom not the slightest evidences of a neuropathic family history could be discovered. The patient himself has, however, been of a very nervous disposition since arriving at manhood. He has always been a very steady smoker, and for six months prior to his first convulsion, which occurred in September, 1876, indulged excessively in his favorite habit, especially on Sundays, upon which day he smoked continuously from morning until night. From the occurrence of the first fit until the patient came under my notice (June, 1878), he had had ten attacks, all of which occurred on Sunday evenings. I placed the patient on fifteen-grain doses of bromide of potassium, and only permitted him to smoke three pipes daily. Under this treatment the fits disappeared until the autumn of 1879, when he had another convulsion. Upon inquiry, however, I discovered that he had discontinued the medicine for a month previously, and had, at the same time, increased his allowance of tobacco. A renewal of the treatment, and a diminution in the amount smoked, has again resulted in a cessation of the attacks until the present time (July, 1880).

CHAPTER III.

PATHOLOGICAL ANATOMY.

THE pathological anatomy of epilepsy is extremely unsatisfactory, and can be disposed of in a very short space of time.

The most varied lesions have been found affecting all parts of the brain, not alone the membranes and substance of the brain, but also the bones of the skull. The latter are usually thickened and the diploë has disappeared, but, in rarer cases, the bones are abnormally thinned. I have found these appearances in the most varied forms of insanity, whether they were complicated with epilepsy or not. Sometimes exostoses are observed in various parts of the skull, and I have reported a well-marked case of this kind in the chapter on etiology. Considerable stress has been laid on stenosis of the foramen magnum, as this anomaly has been observed in quite a number of cases.

Lasegue[1] has recently claimed that epilepsy is due to a malformation or vice of consolidation of the bones which form the base of the skull. According to him, this malformation is indicated by the following characteristic appearances: the frontal protuberance is usually much more marked on the right side than on the left; there is a corresponding projection of the malar bone; the face is rotated, the osseous line of the palate deviates from the median line of the body; deformity of the arch of the palate; lowering or raising of one of the orbits; one side of the face is sunken, corresponding to the projection of the other. In confirmation of his views, Lasegue states that epilepsy usually begins from the twelfth to the eighteenth years, a period which corresponds to that of the consolidation of the bones of the base of the skull.

Garel,[2] who also investigated this topic, found fifty-two cases of facial asymmetry among ninety-four epileptics, or $55\frac{31}{100}$ per cent. Among ninety-four cases of non-epileptics taken indiscriminately, he found thirty-eight cases of facial asymmetry, or $40\frac{42}{100}$ per cent. The difference between epileptics and non-epileptics in this respect is, therefore, too small to entitle the asymmetry to be looked upon as a cause of the disease.

The cerebral meninges sometimes present the evidences of chronic meningitis, and at times they are entirely normal. I have found inflammation of the dura mater (pachymeningitis) in several insane epileptics, and in one of them, who died in the status epilepticus, the membrane on the dura mater was sufficiently thick to produce compression of the frontal and parietal lobes on the right side of the brain. I have, however, found similar pachymeningitic exudations in other insane patients who had never suffered from epilepsy, as well as in sane individuals who have had the most various diseases.

The examinations of the structure of the brain itself has likewise led

---

[1] Bulletin de l'Acad. de Médecine, 1877.
[2] Lyon Médical, Jan. 6, 1878.

to various results. In perhaps the majority of cases no abnormal appearances have been observed. Schroeder van der Kolk,[1] to whose researches great importance has been attached, sums up the results of his investigations as follows :

"In the commencement of epilepsy it would seem that no apparent organic change exists. Rapidly, however, probably in consequence of the repeated congestion, the presence of a more albuminous cellular fluid between the nervous filaments is manifested, which may first cause more or less hardening, and may subsequently give rise to fatty degeneration and softening. In addition, dilatation of the arterial capillaries and thickening of their walls ensue."

"These blood-vessels in the medulla oblongata run chiefly in the region of the roots of the hypoglossus and vagus, as well as in the septum and corpora olivaria. The posterior half of the medulla oblongata from the fourth ventricle, in epileptic subjects, appears, on a transverse section, redder and more hyperæmic than in the normal state, whether the sufferers died during an attack or not."

"In epileptics who bite the tongue during the fit, the capillary vessels are usually wider in the course of the hypoglossus and corpora olivaria ; in those who do not bite the tongue they are wider in the course of the vagus."

Echeverria substantiated the results of Van der Kolk's investigations, and, in addition, found even more advanced lesions. In the medulla oblongata, he observed granular cells, an increase in the number of amyloid bodies, and pigmentation of the ganglion cells, especially in the nuclei of the hypoglossal and pneumogastric nerves ; hyperplasia of the connective tissue was also noticeable. He also observed similar lesions in various portions of the convolutions of the brain, but while they were sometimes absent in these localities, they were invariably present in the medulla.

Echeverria also found the following changes in the cervical sympathetic, which he considers characteristic of the disease : the ganglion cells were broken up, shrunken, or infiltrated with brownish colored granules, which concealed the nuclei.[2] The cells were atrophied from the pressure caused by the increase of slender, nucleated, transparent fibres, fatty granules, and amyloid corpuscles. Many of the nerve-fibres had lost their contents, so that nothing was left but the sheaths ; in others the fibres had become granular ; the tubes, which were destitute of axis cylinders, contained numerous oval nuclei.

Ludwig Mayer, who made careful investigations with regard to these points, has found all these changes in other cerebral diseases, and regards them as secondary to the frequent recurrence of circulatory disturbances. I have made no investigations with regard to the occurrence of such changes in the medulla, but I may state that I have frequently found these lesions in different parts of the cerebral convolutions in various forms of insanity, and have always regarded them in such cases as secondary to chronic cerebral congestion.

It is very evident, therefore, that these lesions are not the causes, but rather the effects of epilepsy.

---

[1] On the Minute Structure and Functions of the Spinal Cord and Medulla Oblongata, and On the Proximate Cause and Rational Treatment of Epilepsy, 1859.

[2] Labimoff states that pigmentation of the ganglion cells is habitually found in older *people, and* is not infrequent even in the young.

Epilepsy may also be secondary to any localized affections in the brain, such as tumors of the meninges or brain tissue, hemorrhages, softening from various causes, abscesses, cysts, etc. Tumors of the dura mater and cortex play an important part in this connection.

There is no doubt, therefore, from this brief résumé of the results of the anatomical investigations with regard to this disease, that there is no pathological anatomy of epilepsy. All the numerous changes which have been observed are either secondary lesions or they shed no light upon the real cause of the affection. We are also inclined to believe that the pathological anatomy of the future will not add much to our knowledge, or rather, will not dispel our ignorance concerning the disease. It appears to us, in view of the perfection to which modern histological methods have arrived, and of the numerous and careful investigations which have been made by enthusiastic observers, that some positive results should have been reached, if they are within the range of possibility. Perhaps it will be left for the physiological chemist, to whose domain so many other pathological questions will be relegated, to discover the true essence of the disease. But our ignorance on this point should not daunt us in the search of a curative remedy. Our art pre-eminently is or should be a healing one, and it is far better to grope blindly and empirically for better therapeutic agents, than it is to fold our arms in despair until the anatomist has informed us what the lesion really is.

Pathology.—Numerous experimental investigations have been made in order to shed some light upon the pathology of epilepsy, but opinions are still at variance on this point. Some authorities locate the primary lesion in the convolutions of the brain, others in the pons and medulla.

Kussmaul and Tenner first showed, by experiments on the lower animals, that anæmia of the brain will give rise to loss of consciousness and general convulsions (at first tonic, then clonic). They concluded, from their experiments, that the primary disturbance was situated in the medulla oblongata (which is now known to be the site of the vaso-motor centre), and that irritation of the medulla gave rise to spasm of the cerebral vessels, anæmia of the organ, and consequently produced loss of consciousness and convulsions. From the result of his anatomical investigations, Schroeder van der Kolk was also led to look upon the medulla oblongata as the site of the disease.

Brown-Séquard has made numerous experiments on the artificial production of epilepsy in animals, especially in guinea-pigs. He showed that the disease develops in these animals after various injuries to the nervous system, such as division of peripheral nerves, incision of the columns of the spinal cord, etc. Within a month to a month and a half after the experiment, the first attack of epilepsy usually develops. After this they may occur spontaneously or upon irritation of the so-called epileptogenic zone, which includes the cheek and anterior portion of the neck. Brown-Séquard also made the curious observation that the young of guinea-pigs, who have been made epileptic in this manner, may develop the disease spontaneously. These experiments have been repeatedly verified by Schiff, Westphal, and numerous other observers. Westphal also showed that similar results may be obtained by striking the animals gently upon the head; in these cases, he found small hemorrhagic extravasations in the medulla oblongata and upper portion of the cervical cord.

Still later, Nothnagel[1] showed that the "convulsive centre" for the

[1] Virchow's Archiv. Bd. XLIV.

muscles of the entire body is situated in the pons varolii. The theory mentioned above was then modified in view of the results of Nothnagel's investigations. This author believes that "the irritation of the vaso-motor centre and of the centre for the muscles (convulsive centre) is co-ordinate," and that one of these parts may alone be irritated. In this manner he explains the variations in the character of the epileptic seizures. Thus, irritation of the convulsive centre alone would give rise to convulsions unattended by loss of consciousness (as in the case mentioned on page 49), while irritation of the vaso-motor centre would produce loss of consciousness or psychical disturbances, according as the secondary cerebral anæmia affects the entire convolutions or only certain portions.

This view presupposes an abnormal irritability (in many cases of a congenital nature) of the pons and medulla.

The English school of investigators, following the lead of J. Hughlings Jackson, have formulated a different hypothesis. Jackson, in an article published in the "West Riding Reports for 1873," states that "defined from the paroxysm, epilepsy is a *sudden*, *excessive*, and *rapid* discharge of gray matter of some *part* of the brain; it is a *local* discharge. To define it from the functional alteration, we say there is in a case of epilepsy gray matter which is so abnormally nourished that it *occasionally* reaches very high tension, and therefore occasionally 'explodes.' The two definitions are different faces of the same thing." Since the publication of this article, Jackson [1] advanced the opinion that the cells of the brain suffer secondarily in epilepsy as a consequence of arterial disease, and that there is thrombosis or embolism of small arteries in most cases.

According to this theory, the nerve-cells are in a condition of unstable equilibrium (attributed by some to excessive, by others to deficient nutrition), and the phenomena of epilepsy are due to the sudden and violent action of these cells, *i. e.*, to the sudden liberation of nerve-force.

The various modifications in the phenomena observed during a paroxysm are supposed to be due to the location of the nerve-cells which are thus affected.

Probably the strongest argument in favor of the theory that the "discharge" begins in the convolutions is found in the fact that so many epileptics present a special sense or even intellectual aura, which could not have been produced by any functional disturbance of the pons varolii or medulla, but must be attributed to a "discharge" of the higher centres, viz., the convolutions.

Gowers claims that the hypothesis of vaso-motor spasm is unnecessary, as all the phenomena can be explained by the discharge of gray matter.

There is no doubt that all the symptoms of epilepsy may be interpreted in the light of Jackson's theory, but his views are, after all, mere matters of speculation, and we shall, therefore, forbear from their further consideration.

---

[1] Lancet, Jan. 25, 1879.

CHAPTER IV.

DIAGNOSIS AND PROGNOSIS.

In chronic cases of grand mal, in which there is a history of repeated typical epileptic convulsions, occurring spontaneously and not accompanied by any other cerebral symptoms, the diagnosis is evident at the first glance. But mistakes are readily made, even in cases of grand mal, when the convulsions occur only at night. Nocturnal incontinence of urine in the adult should always arouse our suspicions in this direction. If, in addition, the patient complains of being worn out in the morning and feeling "as if he had been working all night," if the tongue feels sore and the pillow is stained with a little blood, or if a petechial eruption is found in various portions of the face, especially around the outer angles of the eyes—when all these symptoms are combined, the testimony in favor of nocturnal epilepsy is very strong indeed. In cases of this nature, we should always direct the patient to sleep with a companion, so that we may obtain positive evidence of the existence of the disease. In exceptional cases of this kind we may thus be able to determine the presence of epilepsy in individuals in whom it had not been suspected.

If a patient gives a history that he has suffered some contusion or injury during his sleep and was entirely unaware of any accident, careful examination should also be made. Trousseau mentions a case in which dislocation at the shoulder-joint occurred twice at night, the patient being ignorant of the accident until he awoke in the morning. Basing his opinion on these facts, Trousseau made a diagnosis of nocturnal epilepsy, and other details which were then communicated by the patient dissipated all further doubt.

Sometimes we are called upon to differentiate real from feigned epilepsy. When the malingerer is intelligent and has closely observed the disease, it may be very difficult to detect the simulation. Thus Trousseau relates, in his lecture on epilepsy, that Calmeil simulated an epileptic convulsion so perfectly in Esquirol's presence, that the latter thought the attack was real. Dr. Gorton, of the State Asylum for Insane Criminals, informed me that he had under his care a thief who feigned epilepsy so well that a number of London hospital physicians were deceived. As a rule, however, the deception is readily detected with a little care. An individual feigning epilepsy usually finds a soft spot to fall upon, pallor of the face does not develop, the pupils are not dilated, the reflexes are preserved, the face does not become so dusky during the clonic stage, and the attack is not followed by coma; general sensation is well preserved, and the patient reacts upon a sufficiently powerful stimulus. Voisin regarded the changes in the sphygmographic tracings of the pulse during an attack as pathognomonic, but further investigations have disproved this statement. In determining whether a convulsion is

feigned or real, we should be guided by the *tout ensemble* of the symptoms, and not by the presence or absence of a single one. A great deal will depend upon the quickness of observation and the judgment of the physician.

The diagnosis of petit mal is much more difficult than that of the grand or haut mal. When the two forms are combined, petit mal is readily recognized on account of its combination with well-marked convulsive seizures. But when the former variety alone occurs, its true significance is frequently overlooked, and it is often mistaken for simple syncope or vertigo. Petit mal is often preceded by an aura, and this should always be inquired into very carefully, as it is a very important sign. Furthermore, the attack may occur in a patient who is otherwise in perfect health, and in whom no cause for syncope can be discovered; the former is also often accompanied by inarticulate muttering, and, if the individual is closely watched, slight convulsive twitchings may be noticed in the face or hands.

The sufferer from petit mal is usually unconscious of the occurrence of the "weak spell" and, if not told by those around him, might have a considerable number of attacks without becoming aware of it. Upon close inquiry, we may discover that the patient's memory is failing, that his intellectual powers are not up to their normal condition, and perhaps that another member of the family has suffered from epilepsy or some other severe neurosis.

Quite a large number of cases have come under my observation in which this affection had been entirely overlooked, and we are convinced that the profession is not by any means fully alive to the frequency or gravity of this form of epilepsy. The following case will show how readily the nature of the disease may be misconstrued, as it was only by a mere accident that I was led to recognize its true character.

A young woman, æt. 22 years, consulted me with reference to an angina pectoris which had lasted for four years. She complained of attacks, occurring at irregular intervals, during which she had severe shooting pains in the præcordial region, radiating thence into the left arm; this was accompanied by dyspnœa, great dread, and a feeling as if the heart had stopped beating. At the termination of these symptoms, which only lasted a couple of seconds, the patient would faint away. Upon obtaining this history, I made a careful physical examination of the heart, but the results were entirely negative. I then questioned the patient with regard to the condition of the uterus, thinking that the angina was perhaps the reflex result of irritation of that organ. She gave a history of profuse leucorrhœa, menorrhagia, and severe pain in the back. I then proceeded to make a vaginal examination, but my finger had no sooner come in contact with the os uteri, than the patient became perfectly rigid, and did not answer me when addressed. Upon touching the cornea no response was elicited, the respirations ceased, the pulse remained normal; the color of the face did not change. After a period which appeared to me about a minute, the patient recovered consciousness but was bewildered for a short time. The character of the disease was now evident. The fainting spells were true attacks of petit mal, and the symptoms of angina pectoris merely constituted an aura. Upon questioning the patient more carefully, I then discovered that she frequently had fainting spells without any previous anginal seizures, and that she would fall wherever she happened to be; I also learned that her memory had failed considerably. The only cause to which I could attribute the disease was

mental distress arising from the ill-treatment of herself and mother by her step-father.

The recognition of the variety of peculiar attacks which we have described under the heading of irregular epilepsy is often extremely difficult. Their distinguishing feature consists in the fact that they usually occur in patients who are also affected either with grand or petit mal, and the occurrence of any group of symptoms appearing in paroxysms should always arouse our suspicions under such circumstances. But, as we have previously stated, the usual forms of the disease may never have occurred. Important characteristics of these seizures consist in the fact that they occur suddenly and spontaneously in paroxysms of variable duration; that an aura may be present, that consciousness is either entirely lost or at least disordered, and that no symptoms are noticeable during the intervals between the attacks.

We should always examine carefully into the presence of any neuropathic family history or of any of the predisposing or exciting causes of epilepsy. Whether or no the symptoms described by Griesinger as epileptoid conditions, should be included under this head will depend in great measure upon the individual bias of the observer. Their epileptic character is rendered more probable if the patient has previously suffered from well-marked convulsive seizures. This must be especially taken into consideration before making a diagnosis of epileptic sweating, such as we have referred to on page 57, since very similar attacks frequently occur during what we have termed the menopause neurosis, and evidently stand in no relation to epilepsy

We are frequently called upon to distinguish epilepsy from eclampsia infantum. There is nothing in the symptomatology of the convulsions which will serve to differentiate the two diseases. The convulsions of epilepsy, however, recur at longer or shorter intervals, usually without any exciting cause, after the disease has become developed, and, as a rule, only one convulsion occurs at a time. It is also important to enter carefully into the family history in order to determine whether any hereditary neuropathic tendency is manifested. Eclampsia infantum follows some definite exciting cause, such as dentition, intestinal worms, gastric irritation, or the onset of an acute disease. Very frequently, also, the little patient suffers from a series of convulsions, which follow one another in rapid succession, and which may last for hours with hardly any intermission. In not a few cases, however, it will be impossible to make a differential diagnosis for a very long time, and in some instances this difficulty is obviated by saying that eclampsia infantum, if frequently repeated, may become converted into true epilepsy.

Difficulty is sometimes experienced in distinguishing the uræmic convulsions of cirrhosis of the kidneys from true epilepsy. The uræmic convulsions may occur suddenly in a patient who has apparently enjoyed perfect health, and indeed several series of attacks may be repeated at variable periods, although the patient appears to be well during the intervals. There are several symptoms, however, presented by patients suffering from granular kidneys, which are very characteristic and enable us, with a little care, to make a correct diagnosis. In the first place, various grades of hypertrophy of the left ventricle are present in all cases; this usually produces no symptoms and is unnoticed by the patient. The tension of the arteries throughout the body is greater than normal, and is shown by increased resistance of the radial pulse. The urine is increased in quantity and diminished in specific gravity. There may be a slight

amount of albumen present, or the urine may be entirely free from it; a few hyaline casts are observed from time to time. The character of the convulsion also differs somewhat from that of epilepsy. It is usually very violent, is never preceded by an aura, and the subsequent coma may be very profound and even give rise to serious alarm. Furthermore, we not unfrequently find that the convulsions occur in series, during which cerebral hemorrhage may be produced.

Hysterical convulsions are usually distinguished with readiness from those occurring in epilepsy. The former generally occur in young females, who also present other well-marked symptoms of hysteria; consciousness is not lost during the attack, but is, at most, slightly disordered; the convulsive movements do not present the same regularity observed in an epileptic seizure; the patient throws herself into peculiar positions, rolls around from one place to another; respiration is not so completely interrupted, and the entire attack may last from several minutes to an hour or more.

Hysterical convulsions may be very frequently repeated, but coma does not often develop after these attacks. Bourneville has shown that a rapid succession of hysterical seizures may be readily diagnosticated from the status epilepticus (vide page 51), by the fact that the latter is attended by a rapid rise of temperature, while the former is never accompanied by this symptom.[1] Finally, hysterical convulsions may often be arrested by measures which are directed toward the imagination of the patients. We must not forget, however, that epilepsy and hysteria are frequently combined in the same patient, especially among the inmates of an hospital for epileptics.

The epileptic seizures which occur as a symptom of cerebral syphilis (gummata, vessel changes, etc.), should also be distinguished from idiopathic epilepsy. In the former disease, the patient generally gives a history of previous infection with the syphilitic virus, and other manifestations of the cerebral disorder are presented prior to the development of the epilepsy. Prominent among these symptoms is intense headache, which always grows much worse at night; ptosis, pupillary disturbances, or paresis of various portions of the body make their appearance; optic neuritis is often present. When convulsions occur, any paresis which may have been present is usually deepened into paralysis, and this may partially disappear after a variable period. Not infrequently the convulsions are at first unilateral, and we have sometimes seen them confined to one limb. Unless active treatment is instituted, however, the convulsions soon become general, and then differ in no respect from those of ordinary epilepsy. One of the most characteristic features of this disease is its curability under anti-syphilitic remedies, though, unless treatment is begun soon after the first cerebral symptoms have presented themselves, there is great danger of a relapse. It is also important to remember that these manifestations of syphilis generally make their appearance after the age of thirty, so that this fact should always put us on our guard.

Epilepsy offers, on the whole, a very gloomy prognosis, and some physicians have even gone so far as to deny its curability in any case. This is, however, not true, as a very small percentage of cases appear to recover spontaneously.

---

[1] As previously mentioned, I have observed a case of status epilepticus in which *no rise of* temperature occurred.

It must be remembered that the convulsions may run a very irregular course, and a case should, therefore, not be regarded as permanently cured unless at least three years have elapsed since an epileptic seizure has occurred.

In a considerable number, perhaps a majority, of the cases, the frequency and severity of the fits may be very materially diminished, and in a few they will entirely disappear. The chances of recovery increase according as the disease is due to some definite, removable cause, as in certain cases of reflex epilepsy. The duration of the disease prior to beginning treatment is also of importance, the chances of recovery being greater the earlier the affection is recognized and treated; it is generally supposed that, if the patient has already had 100 convulsions before the treatment is begun, the prognosis is very poor; if as many as 500 have occurred, there is not the slightest chance of recovery.

The prognosis is somewhat better when the disease begins early in life, than if it develops during manhood. An hereditary influence decidedly lessens the patient's chances, and cases of this kind are also more liable to become complicated with mental disorders. It is still undecided whether the grand or petit mal is the more curable. Judging from my own experience, I am inclined to believe that the latter form is more susceptible of improvement than the former. I have also begun to entertain considerable doubt in the general belief that petit mal is more apt to be attended with psychical disorders than the grand mal.

Those patients in whom epileptic mania has developed usually go from bad to worse, and generally terminate eventually in dementia. Nevertheless, I have seen several cases in the young in whom a return to a normal condition of intellect was effected, although the patients had suffered from considerable mental disturbance.

The prognosis as regards life is excellent, and death only occurs in exceptional cases from an accident during the convulsion, such as suffocation, drowning, fracture of the skull, cerebral hemorrhage, etc. When the status epilepticus develops, however, a fatal termination is not infrequent either in the convulsive or meningitic stage. But these cases are comparatively rare.

In some cases, an example of which is shown in the following history, a fatal result follows, although the cause of death remains unknown or is very obscure:

CASE XIII.—Anne C., æt. 26 years, single, no hereditary taint discoverable. The patient was always of a nervous temperament, but in tolerably fair health, until five years ago. At that time she obtained extremely little sleep for a period of five months (was acting as a nurse). The first epileptic paroxysm developed shortly afterward, and was of the nature of grand mal. The attacks then made their appearance with continually increasing frequency, the memory began to be impaired, and, finally, the patient became very forgetful. When she first came under my observation, a marked hysterical condition was manifest. The treatment consisted of the administration of bromide of potassium, gr. xv. t.i.d. As the patient was unable to find employment on account of her condition, I admitted her to my wards in Randall's Island Hospital, February 14, 1880, and the dose of bromide was then increased to gr. xxx. t.i.d. Soon after admission, the hysterical symptoms became aggravated, and the patient (who was a Protestant) began to suffer from the delusion that the nurse and the Catholic patients in the ward were abusing and persecuting her.

At intervals she refused food, stating that an attempt was being made to poison her. On several occasions she attempted to run out of the hospital in her night-gown, and sometimes used violence toward the other patients. She gradually became more morose and listless and finally took to bed (March 20th). After this she lay in a stupid condition, interrupted on one occasion by an attack of excitement, during which she attempted to get out of bed, and struck an attendant. She then refused food during an entire week (for fear of being poisoned), and was fed by means of rectal enemata. The patient gradually sank and died April 8, 1880.

Autopsy—held by the curator, Dr. Habirshaw. *Brain.*—On the inferior portion of the right occipital lobe was a small patch of enlarged vessels, presenting the appearance of minute hemorrhages. The pia mater was slightly roughened over the vertex and adherent in some places; the interior of the brain, including the ganglia, was congested. *Lungs.*—The lower lobes of both lungs contained a few lobules of catarrhal pneumonia of various sizes, attended with hypostatic congestion; some of the bronchi contained thick pus.

Neither the morbid appearances in the brain or lungs were sufficient to account for the symptoms. Another case of a very similar nature, and also attended with a fatal result, has come under my observation. In this instance I was unable to obtain an autopsy.

CHAPTER V.

TREATMENT.

MANY authorities regard the disease as entirely incurable, and do not even attempt to treat it, while others have recorded a large proportion of successes. In our opinion, the truth lies midway between these two extremes.

One of the most important points to be remembered is that every case must be treated by itself; that we must treat the patient, not the disease. In pursuance of this object, we should, if possible, ascertain the etiology of the affection. In a large proportion of cases this is impossible, and in others the cause cannot be removed, even when known. But in a certain number, especially in reflex epilepsy, the cause cannot only be determined but can also be readily made to disappear. It is unnecessary for me to refer again to these etiological factors, as they have been discussed in extenso in the section on etiology, and their treatment belongs partly to surgery, partly to other branches of medicine. The treatment of diseases of the ear, throat, chest, genital organs, etc., which act as causes of the epileptic seizure, is similar to that usually resorted to in such affections. The prominence which has been attached to trephining for injuries of the skull which have acted as causes of epilepsy, requires that we should enter into this subject somewhat more fully. There is no doubt that the advocates of trephining for epilepsy have entertained exaggerated views with regard to the influence of injuries of the skull, in the production of the disease. Among 3,000 cases of epilepsy, Althaus did not find a single one which presented fracture of the cranial bones, with depression of the skull. Several cases have come under my notice in which the disease was supposed to be due to injury of the skull, although careful inquiry showed no grounds for this assertion. As we have before remarked, the statements of patients or their relatives with reference to etiology should always be subjected to careful scrutiny before being accepted. In examining the favorable statistics with regard to the effects of trephining which have been advanced by several writers, we must take into consideration the fact that, as a rule, the unfavorable cases are not reported, and furthermore, that a considerable proportion of the favorable ones have been published within a few months after the operation, at a period, therefore, in which it is unwarrantable to draw any conclusions with regard to the final result.

On the whole, therefore, we should not advise the performance of trephining, unless the epilepsy evidently followed an injury to the skull, which was accompanied by distinct fracture of the bones, with depression. If irritation of the injured part is capable of producing a convulsion, the indications for the operation are rendered much stronger.

In cases of syphilitic epilepsy, the indications for treatment are very clear. When the convulsions occur during the early secondary stage,

as in the form recently described by Fournier, mercurial treatment is alone required. When the attacks occur during the tertiary stage, and are either *sine materia*, or form part symptom of other manifestations of cerebral syphilis, iodide of potassium should also be administered in combination with mercury. If the disease has not lasted too long, the prognosis is very good, but we cannot afford to use the drug in homœopathic doses. I usually administer the mercurial separately from the iodide, as the latter must be increased very much more rapidly than the former. The iodide of potassium is first given in fifteen-grain doses three times a day, from one-twenty-fourth to one-sixteenth of a grain of the bichloride of mercury being exhibited at the same time. In a week, the quantity of iodide may be increased by half, and if a fit has occurred in the interim, the dose may be doubled. The quantity is gradually increased in this manner until the iodide eruption makes its appearance or the stomach begins to revolt. If this quantity succeeds in preventing the occurrence of the fits, the amount is slightly reduced, and the patient held at this dose. If the attacks continue, the quantity of iodide administered should be increased until, if necessary, half an ounce, or even an ounce, is taken per diem. When such large amounts are administered, I usually order thirty grains of subnitrate of bismuth, with five or ten grains of the bicarbonate of soda, to be taken before each dose of the iodide, in order to counteract the irritating effects of the latter upon the gastric mucous membrane. These patients often suffer from violent headache. In such cases, frequently repeated blisters, applied to the nape of the neck, prove very serviceable; in others resort may be had to the use of the actual cautery, but I have not derived so much benefit from this measure as from the application of blisters.

One great drawback against which we have to contend in these patients is owing to the fact that they are apt to discontinue the remedy as soon as the fits have ceased for a couple of months. This is a great mistake, and I always make it a rule to forcibly impress upon my patient the necessity of continuing treatment for a year and a half or even two years after the last fit occurred. A patient who has, at any time, had manifestations of cerebral syphilis, is always liable to a relapse, and this can only be prevented by the long-continued and faithful use of the iodide. We must also bear in mind that the quantity to be administered is not regulated by the dose laid down in the materia medica, but by the effect produced upon the patient.

When the attacks are due to excessive sexual intercourse, or to masturbation, such practices must, of course, be interdicted. But while the former cause can be easily removed in the majority of cases, the latter desideratum is not so readily obtained. If the masturbator, usually a child, has a narrow prepuce which is causing irritation of the glans, it should be circumcised, and I have recently had a case in which the fits have remained absent for the last four months since the operation, no other treatment having been adopted. In girls, in whom no local treatment can be employed, we can only resort to careful watching of the patient by the relatives. I also usually recommend that the patient be compelled to sleep with an adult, as I have found that many of the little patients will masturbate as soon as they get warm in bed. In those rare cases in which the attacks develop during coitus, the performance of the sexual act must, of course, be strictly prohibited.

But, as we have shown in the chapter on etiology, in the majority of *cases* the cause is either unknown or is of such a nature that it cannot

be relieved either by surgical or medical measures. We must also remember that a considerable proportion of the cases of reflex epilepsy only come to us for treatment several years after the cause first began to operate, and that, in many of these patients, the epilepsy has acquired an independent existence, and will not disappear, even though the original cause be successfully removed. In the large majority of cases, therefore, we can only treat the disease symptomatically.

The treatment of epilepsy may be divided into two parts, viz.: the use of general and medicinal remedies. We shall first study the former.

There is a widespread notion in the profession, as well as among the laity, that epileptics must be kept idle; that they should not be allowed to do either any mental or physical labor. This view is undoubtedly erroneous. With regard to mental exercise, there is no doubt that the tendency of the disease itself is to produce deterioration of intellect, and this mental infirmity will be increased by allowing the patients to grow up uneducated. It is true, however, that excessive mental work will aggravate the frequency of the occurrence of the fits. In fact there are some patients who only have a convulsion after they have undergone some severe or prolonged mental strain. Continued reading is especially injurious to epileptics. But we must not fall into the other extreme, and debar them from all reading. It is, indeed, not alone not injurious to allow them to read, attend lectures, amusements, etc., but it is even beneficial when confined within proper limits. Such a course of conduct tends to preserve the mental tone of the patient and to divert his attention from his malady, which is, otherwise, always uppermost in his mind.

This practice also tends to prevent the patient from continuing the habit of masturbation, which, when it once gains the upperhand in an epileptic, has a bad influence upon the frequency of the attacks.

These remarks will also apply to manual labor; the patients usually do well with a proper amount of physical exercise. Of course this must not be excessive. In some of my dispensary patients I have frequently seen the fits increased in frequency to a terrible extent, and, upon inquiry, have found that this could be attributed to overwork, and that their number immediately diminished when the character of the work was changed.

When patients, whether male or female, have become epileptic at the age of puberty, marriage is frequently advised by the physician in attendance. The theory on which this advice is based, appears to be that the epilepsy is due to some unknown derangement of the sexual system, and that this will disapppear in consequence of the regular performance of the sexual act. A few cases are on record which appear to substantiate this view, but they are very exceptional. It must also be remembered that, even in some of these cases, although the epilepsy disappeared in the parent, it reappeared in the offspring.

But the majority of writers are opposed to the marriage of epileptics. In Denmark this matter has become the subject of legislative interference, and the fact that one party to a marriage was an epileptic prior to the marriage, without the knowledge of the other, constitutes a valid ground for divorce. We believe that this is, theoretically, the proper plan to pursue. Even if we hold that marriage will prove curative of the affection, the risk which the parent runs of transmitting this terrible disease to the offspring is so great that he should be warned against marriage. Although there is no legislation on the subject in this country, the medical profession can do a great deal toward preventing such alli-

ances. In the event that our advice in this respect is disregarded, as it so frequently is, it becomes a grave question whether it is not our moral duty to override the letter of the code, and inform the other partner in the projected marriage of the actual state of affairs.

If the general condition of the patient is below par, the ordinary tonic remedies, such as iron, quinine, cod-liver oil, etc., should be administered. Some authors have discountenanced the use of iron in this disease, because it tends to produce plethora. In order to test the truth of this statement, I have often given iron, not alone to anæmic epileptics, but also to those who were in excellent general health. I have yet to find any bad effects from its administration.

Opinions vary with regard to the diet of epileptics. The notion has become prevalent among the laity, and is even widespread among physicians, that nitrogenized food should be sparingly partaken.

Thus, Hughlings Jackson advises that epileptics should not eat much nitrogenized food, nor, indeed, much of any kind of food, basing his opinion on the view that the nervous tissues in this disease are over-nourished with regard to quantity.

Dr. Merson,[1] who made some extended observations on this question, arrived at the following conclusions: "In a considerable number of those who took nitrogeneous food during the first month, it was observed that soon after commencing that diet they became much more dull and stupid than they had previously been, would sit in a dreamy, listless manner for a great part of the day, were very slow and languid in their movements, and took little notice of what was going on around them. . . . . No relation was traced between it and the recurrence of the fits. As soon as the diet was changed to the farinaceous, it was remarked that the condition of hebetude began to pass off, and in some of the cases the change was very remarkable. The improvement in the mental condition was not always accompanied by any marked diminution in the number of fits." Merson continued his experiments for two months, and found a slight decrease in the number of fits in a large proportion of the patients who were kept on a farinaceous diet, but the difference was not very marked.

The results obtained by this experimenter are not decided enough to be very convincing. My own experience leads me to believe that it is unnecessary to limit the quantity or quality of the food, with the exception that all heavy, indigestible substances are to be avoided, as well as strong tea or coffee. The use of spirituous liquors should also be carefully interdicted.

We now turn to the medicinal agents which are employed to combat the disease itself. The number of drugs which have been used in the treatment of epilepsy is legion, and a couple of pages could be filled with their mere enumeration. I shall, however, only mention those which have proven useful in my own practice. In considering the remedial effects of any agent in this disease, we must bear in mind that any change is apt to prove beneficial for a time. Thus, if the administration of one drug is stopped and another is substituted for it, the number of fits will be frequently diminished for several weeks or even months, after which they resume their former frequency.

Bromide of potassium is by far the most potent of all remedies. Various other bromides (sodium, ammonium, etc.) have been employed

[1] West Riding Lunatic Asylum, Med. Rep. V., 1875.

in preference, but I have never seen them succeed where the potash salt failed. It produces a considerable amount of improvement in the majority of cases, in others it is entirely useless, and in a few its administration must be discontinued, as the patients grow steadily worse. I have found that the latter event is more apt to occur in cases of nocturnal epilepsy or in petit mal. The character of the fits often changes under the influence of this drug. As an indication of improvement, we sometimes find that the aura alone develops at times, the convulsions remaining absent. Exceptionally, I have also noticed that in cases in which the convulsions had previously occurred without an aura, the latter symptom developed under the influence of the bromides. The mental symptoms also undergo improvement; the irritable temperament may disappear, the dull, stupid expression vanishes, and the patients may acquire a greater interest in the affairs going on around them.

The manner in which the drug is administered must be carefully regulated. When benefit is derived from this remedy it should be continued uninterruptedly for years, and I always make it a rule to administer it for at least two years after the last convulsion has occurred. The initial dose is fifteen grains, t.i.d., taken preferably on an empty stomach (about three hours after meals), and in plenty of water. When the stomach is dainty, it may be combined with a little bicarbonate of soda and a simple bitter. Dr. E. C. Seguin advises its administration in Vichy water, and I can also recommend this plan as very satisfactory. In cases of nocturnal epilepsy a double dose should be taken at night. The quantity administered should be gradually increased until slight bromism is produced (irritability of the stomach, acne eruption, anæsthesia of the pharynx and velum palati, fetid breath, feeble pulse, drowsiness). Different patients vary greatly in this respect, and I have sometimes given three drachms daily for several weeks at a time before any symptoms of bromism became apparent.[1] When these symptoms develop, the quantity administered must be diminished, or the drug entirely withheld for a few days. My own experience is corroborative of that of Dr. Seguin who found that the acne-eruption of bromism may be held somewhat in check by the administration of small doses of Fowler's solution. After the bromism has subsided the remedy is continued in somewhat smaller doses for a few months, at least, before it is renounced as useless. The patients should be strongly impressed with the idea that, under no consideration, should the bromide be discontinued, unless under the advice of the physician. If patients, who formerly had a number of fits per week, go several months without a convulsion, they often consider themselves cured, and omit the medicine. We often find, in such cases, that the fits return after a very short intermission. When very large doses are required it is sometimes advantageous to combine the bromides with hydrate of chloral, ten to fifteen grains of the latter being given at a time. In this manner we can diminish the amount of bromide to the point of tolerance by the patient, and, in some cases, the chloral appears to have a special remedial action. This plan should not be continued, however, for a very long time, as hydrate of chloral soon exerts a deleterious influence on the general health.

Belladonna was highly praised by Trousseau in the treatment of this

[1] Three cases have come to my notice in this city, in which death was due to overdoses of bromide of potassium; the condition of the patients should therefore be very carefully watched when large doses are given.

disease, and other observers have also reported favorable results from the use of this remedy. At the present time atropia is used in preference, as the alkaloid is much more constant in its effects, and the dose can therefore be graduated more carefully. My experience with it in diurnal attacks of grand mal has not been very satisfactory; at the most I have merely noticed a slight diminution in the frequency of the convulsions. In nocturnal epilepsy, however, and especially in the various forms of petit mal, I have derived marked benefit from this drug. It should be begun in doses of $\frac{1}{96}$ grain, repeated three times a day, and gradually increased until dilatation of the pupils occurs; it should not be pushed beyond this slight physiological effect. As an example of the rapid improvement which sometimes occurs, I may refer to the patient mentioned on page 52, in whom the disease had lasted for four years before coming under treatment, and who had three or four attacks of petit mal daily. As soon as the patient was brought under the influence of atropine the attacks disappeared, and during the entire period in which she was under observation (upward of a year), no attacks occurred unless the drug had been discontinued for a few days. The memory also improved very rapidly.

Strychnia has been very little used in epilepsy, but I have sometimes obtained excellent results in cases similar to those in which I employ atropine, viz., in nocturnal attacks and in petit mal, especially in the former. In such cases it is sometimes combined to advantage with bromide of potassium. The dose varies from one-forty-eighth to one-thirty-second of a grain three times a day, and continued uninterruptedly so long as it produces good results. It possesses the advantage of acting as a nerve tonic, and may be continued for a long time without producing any deleterious effects on the economy. Conium is sometimes used in this disease, but I have usually given it in combination with bromide of potassium, when the latter does not give sufficiently good effects. The dose is five drops of the fluid extract, gradually increased. The remedy must be exhibited very cautiously, as some patients are very susceptible to its influence, and it readily produces symptoms of poisoning.

Quite a number of metallic remedies have been employed from time to time, but we shall content ourselves with mentioning two, viz.: nitrate of silver and oxide of zinc. Nitrate of silver for a long time occupied a prominent part in the treatment of epilepsy, and some undoubted cases of recovery have occurred under its use. At the present time, however, it is very rarely administered, and this is undoubtedly due, in part, to the fact that patients are occasionally met whose entire integument has become blue from the long-continued use of the drug, while the epileptic convulsions have persisted with all their original severity and frequency.

Oxide of zinc appears to be gaining more favor recently than it formerly enjoyed. It is employed either alone or in combination with bromide of potassium, in doses of five to ten grains, which may be gradually increased to twenty grains. I have seen good effects from it when given in both ways, but do not think that it can compare with the bromides in efficacy.

In those cases, however, in which the bromides are useless, or the other remedies mentioned above are not indicated, faithful trial should be made of the zinc salt. Valerianate of zinc has also been recommended, but it possesses no advantage over the oxide; in addition, it is extremely disagreeable to the task.

Nitrite of amyl has recently come into vogue in the treatment of

epilepsy, especially when the convulsions are preceded by an aura. It should always be used whenever the warning is sufficiently long to enable the patient to inhale it before the fit begins. The dose varies from two to five drops (by inhalation), and even more may be required, as some patients become very quickly habituated to its effects. A convenient plan consists in keeping a single dose of the amyl in thin capsules of glass, which can be carried in the pocket, and crushed in the fingers as required.[1] If the patient is too poor to afford this expense, he may keep a dose of the remedy in a small glass vial, and, when he feels the fit coming on, can pour the amyl into the palm of his hand and then inhale it. By some physicians it is administered regularly in three to five drop doses, t.i.d.; I have tried this plan in a few cases, but have not derived any benefit from it. When given during the aura, however, it very frequently aborts the attack, and in this manner may very decidedly reduce the number of convulsions. Some of the patients complain that when a fit does occur under such circumstances it is more than usually severe.

Nitrite of amyl has also proved of decided advantage in the treatment of the status epilepticus, and, in fact, appears to be the only agent which promises any chances of success in this complication. In these cases, however, much larger doses are required, and as many as ten to fifteen drops are often necessary.

Galvanism has recently been employed in this disease, but the majority of observers have come to the conclusion that it is entirely useless.

---

[1] This plan was devised, I believe, by Dr. McBride of this city.

NEURALGIA.

CHAPTER I.

CLINICAL HISTORY.

NEURALGIA is a paroxysmal disease, and is chiefly characterized (sometimes solely) by the presence of pain. This appears under peculiar conditions which distinguish it from other painful affections. The paroxysm usually begins quite suddenly, although it is sometimes preceded for a little while by sensations of cold, numbness, or "drawing" in the affected region. Then suddenly a twinge is felt along the course of one of the nerves, and instantly subsides, to be followed in a few moments by another, and then by another, the intervals between the pains growing gradually or rapidly shorter, until finally they may become almost continuous. Their character is variously described by different patients, sometimes as shooting, darting, lancinating, boring, twisting, wrenching, "as if a coal of fire were being drawn along the nerve," etc. The pains are sometimes truly atrocious, and have frequently led patients to commit suicide.

The paroxysm develops spontaneously, or in consequence of some very trifling cause, such as a movement of the lips or cheeks while speaking or eating, in trigeminal neuralgia, or a slight movement of the foot, in sciatica, etc. The pain always runs along the anatomical course of certain nerves, and usually in the direction from the centre to the periphery, although the reverse of this statement sometimes holds good. I have especially observed the latter state of affairs in brachial neuralgias, but I am unable to explain the reason of this peculiarity. Another striking characteristic of a large number of neuralgic affections is their periodicity. In those which are due to malarial influences this is the rule, and the paroxysm of pain occurs with the same regularity as the chill of intermittent fever; it usually assumes the quotidian, sometimes the tertian type, but it has never, in my experience, occured at longer intervals than on alternate days. But periodicity is frequently manifested even in cases which are not connected with malaria. I have noticed, however, that the paroxysms are more apt to occur in such cases toward the middle or latter part of the afternoon, differing in this respect from the former variety. Periodicity may even be noticeable when the neuralgia is due to an organic affection of the nerves, and I saw it so well marked in the first stages of a case of cancer of the vertebræ that a distinguished physician of this city, who saw the patient in consultation, at first made a diagnosis of malarial neuralgia. The subsequent history of the patient, however, showed the incorrectness

of the diagnosis, and the case finally went on to a fatal termination. This peculiar character of periodicity is not restricted to neuralgia, but is also common to other nervous affections, and no very satisfactory explanation has ever been offered for its occurrence. The duration of the paroxysms is extremely various; sometimes they only last a minute or two, sometimes they may continue for several days or even longer, with very short intermissions. As a rule, the duration of the paroxysms increases with the progress of the disease, and those which occur in old age are usually the most violent and prolonged.

The other sensory disturbances include the *puncta dolorosa* or painful points described by Valleix, and which were considered by him as characteristic of neuralgia. This term refers to circumscribed spots, situated along the course of the nerves, where they emerge from bony canals or foramina, where they pass through firm aponeuroses, or where they become superficial under the skin, and sometimes under the mucous membranes. Pressure upon these localities during an attack of neuralgia produces severe pain at the site of pressure, and may also reproduce the pain in the corresponding nerve. The painful points are sometimes observed during the intervals between the paroxysms, though not by any means so often as during the attacks. If the skin is pinched up in a fold over the site of the painful point, and pressure is made upon the integument, we can readily determine that the pain is not due to hyperæsthesia of the skin. In fact, the reverse is sometimes noticed, and the integument is found to be decidedly anæsthetic, while the underlying nerve is exquisitely tender to the touch. Valleix stated that the *puncta dolorosa* were present in almost all cases of neuralgia, but, in common with a great number of other observers, I have found them absent in a large percentage of cases. It would, therefore, be unwise to exclude the diagnosis of neuralgia from the absence of the painful points.

Trousseau, in combating Valleix's view of the pathognomonic character of this symptom, called attention to the presence of the *point apophysaire*, or spinal point, as characteristic of neuralgia. He states that "the spinous point, as its name indicates, is situated over the spinous processes of the vertebræ, and, since my attention has been drawn to it, I have never known it to be absent." The spinous point usually corresponds to that vertebra from which the affected nerve emerges, but it is a remarkable circumstance that a tender spot is sometimes felt upon pressure over the first or second cervical vertebræ in cases of trigeminal neuralgia. I have observed this in quite a number of instances, and, in some have succeeded in sending a thrill of pain through the affected fifth nerve by pressing upon the spinous processes of the tender vertebræ. At times pain is not produced by pressing upon this part itself, but will develop when pressure is made immediately to one side. In such cases, I have always found that the painful spot was situated upon the same side as the neuralgia. But these "spinous points" are not by any means so frequent as Trousseau believed, and, in fact, great caution must be exercised in determining their presence, especially in females. We should never rest satisfied with one examination, but should press upon the vertebræ several times in succession, and at the same time endeavor to divert the attention of the patient from the object of the examination. If one of the processes is really tender, pressure upon it will produce a change in the expression of the face, which is more reliable than the mere statement of the patient. If we pay attention to all these details, we *will soon come* to the conclusion that a large proportion of the spinous

points are purely imaginary. Although we do not attach the same diagnostic importance to the *points apophysaires* as was attributed to them by Trousseau, they possess considerable importance with regard to therapeutics, as we will endeavor to show in the chapter on treatment.

Hyperæsthesia and anæsthesia in the distribution of the affected nerves are frequently observed. They were first described by Tuerck, in 1850, and later by Trousseau, who thought that the presence of anæsthesia was indicative of deep-seated nerve lesions. Nothnagel[1] has studied this subject very thoroughly, and finds that hyperæsthesia is primary, and lasts from two to eight weeks, and is then followed by anæsthesia. Erb's observations corroborate those of Nothnagel to a great extent, but the former author has also determined the existence of anæsthesia in the first week of the disease; in some cases no sensory disturbances have been noted. In a few instances the hyperæsthesia or anæsthesia were diffused over the entire corresponding half of the body.

Attention must also be drawn, at this point, to the frequent presence of irradiated pains in other parts of the body. Thus it is not at all infrequent that, at the height of a paroxysm of trigeminal neuralgia, pain is felt in the occipitalis major nerve on the same side; the development of trigeminal neuralgia during the course of cervico-occipital neuralgia is observed with less frequency. At times, however, the irradiation may occur to remote parts of the body, as is observed in the development of trigeminal pain during the course of sciatica. The irradiated pains are usually not so severe as those of the primary paroxysm, and they only develop when the latter attains considerable intensity.

In a large percentage of cases the symptoms which we have described above are the only ones observed, and they may be unaccompanied by any others during the whole course of the affection. But severer cases, and sometimes even milder ones, may be accompanied by various motor, vaso-motor, secretory, and trophic disturbances, which may prove much more serious than the primary affection.

We will find, during our consideration of special neuralgias, that while the motor, vaso-motor, and secretory complication are much more frequent than the trophic disorders, the latter are more important, and will also merit careful attention.

Motor complications.—When the neuralgia affects purely sensory branches of a nerve, the motor complications are necessarily reflex; when the nerve is mixed, they are usually manifested in the course of the same nerve, though even in the latter instance they may be radiated to other branches.

The reflex complications are best seen in *tic douloureux*, in which the muscular twitchings, induced by the neuralgia of the fifth pair, appear in the facial muscles which are supplied by the seventh. They vary from slight fibrillary twitchings to well-marked and rapid convulsive movements. The convulsive phenomena occurring in the course of the affected nerve are best observed in sciatica, and I have sometimes seen the limb raised violently from the ground in the course of this affection. Paralyses have also been noticed during neuralgias, but although they have been described during the first stages, I have never observed them until the neuralgia has lasted for a long time. The paralysis is, of course, most marked in the limbs, and I have seen considerable loss of power in inveterate sciatica. This can be readily distinguished from the immobility

[1] Virch. Arch. Bd. 54, 1872.

of the parts caused by a dread of the renewal of the pain on motion, and in my experience has always been attended with considerable atrophy of the muscles—an atrophy which I could not entirely explain on the theory of the disuse of the muscles, and which I was therefore compelled to regard as a trophic change.

Vaso-motor complications.—These are not very numerous or important. During the paroxysms the arteries leading to the affected region are frequently dilated and pulsate strongly and visibly; the surface of the body is red and hot, and its temperature is somewhat raised when compared with the corresponding part of the body on the opposite side. In other cases, on the contrary, the reverse is observed, the skin is pale and cool, and slight rigors are experienced in the affected region. A certain amount of œdema of the subcutaneous cellular tissue may develop during a paroxysm, but it is never very marked, and usually disappears shortly after the termination of the attack.

Secretory complications.—These are observed almost exclusively in trigeminal neuralgia, because this is almost the only form in which a nerve is affected which possesses an influence over any of the secretions.

For this reason we prefer to postpone their discussion until the consideration of the special varieties of neuralgia.

Trophic complications.—We now enter upon an extremely interesting field of observation, which is at the same time of great importance, since the effects produced are frequently of a lasting character. This subject also opens up the question of the existence of special trophic centres, but the character of this article precludes our entering into the discussion. We will therefore assume, without further argument, that the entire series of changes which we shall describe in this section are due to an affection of special trophic nerves. And we shall first take into consideration the trophic affections of the skin.

We must premise our remarks by the statement that the severest forms of cutaneous trophic disturbances (glossy skin, deep ulcerative eruptions, etc.) only occur when the nerves are seriously injured or inflamed, and therefore rarely form part of the clinical history of simple neuralgia.

The lesions of the latter include simple atrophy of the entire skin, which appears smoother than that of the corresponding part of the body, is thinner than on the opposite side, and appears to be more shining than normal. In one case I also observed lesions of an opposite nature, viz., hypertrophy of the skin, a phenomenon which I have found referred to by a few authors, but described by none. The patient in question was suffering from neuralgia of all three branches of the trigeminus (the motor branch was also involved, and the case will be again referred to in the chapter on peripheral paralysis). Upon first examining him, I thought that he was suffering from facial paralysis on the affected side, as the face remained almost motionless on that side, and the natural folds of the skin were partially effaced. Upon careful investigation, however, I found that this condition of affairs was due to hypertrophy of the skin (perhaps, also, in part, of the subcutaneous cellular tissue), which was dark, rough, and could with difficulty be pinched up into a fold. The apparent facial paralysis was due to the inability of the muscles to move the thick and stiff integument, although there was no reason to believe that the muscles had lost their power.

The skin in severe neuralgia is also apt to take on an erysipelatous action, which presents the appearances of ordinary erysipelas, but does

not run a severe course, and never endangers the life of the patient. Pigmentation and roughening of the skin have been noticed by several observers, notably by Anstie; it sometimes disappears after the paroxysm has subsided. In one case I saw a scaly eruption develop in the course of the affected nerve, and disappear as the neuralgia was relieved. Herpes zoster constitutes one of the most interesting of these trophic affections of the skin. This is observed almost exclusively in trigeminal and intercostal neuralgias, especially in the latter, appearing sometimes in severe and sometimes in the course of mild affections.

The eruption appears usually on the right side of the body, and is strictly limited to the course of the nerves; in very rare instances it appears on both sides at the same time, and, if it is situated along corresponding intercostal nerves, may form a complete zone around the trunk. The eruption consists of large vesicles, situated on an inflamed base; they contain, at first, a clear, limpid fluid which, at a later period, becomes cloudy and opaque; the vesicle usually ruptures and a scab forms, which gradually dries and disappears. Sometimes the eruption appears before the neuralgia, but usually the reverse is noticed. This eruption may present great importance on account of the peculiarity of its site; we shall return to this phase of the subject in discussing trigeminal neuralgia.

The appendages of the skin may also become involved in these trophic changes. The hair frequently changes its color during the paroxysms, and is restored during the intervals. One of my patients, a woman suffering from supra-orbital and occipital neuralgia, noticed, during each paroxysm, that a lock of hair in the course of the supra-orbital, and another in the course of the occipitalis major nerve, turned gray, but that the original black color was restored after the paroxysm had subsided. If this process is frequently repeated, the hair may finally remain permanently gray. In other instances, the hair acquires a coarser and more brittle texture (Anstie). In some cases the hairs have a tendency to fall out (especially in neuralgias of the nerves of the limbs), in others a denser growth develops in the course of the affected nerves. The nails may become pale and discolored, and marked by irregular, transverse furrows; they are also apt to be clubbed and to have a diminished rate of growth. Under such circumstances they present a similar appearance to that observed in certain cases of cerebral hemiplegia which are attended with trophic changes in the joints, fingers, and toes. Traumatic neuralgias are complicated by certain other more profound trophic changes of the skin, to which we shall refer at a later period.

The muscles may undergo atrophy quite rapidly, and this is undoubtedly a trophic change, though some authorities consider it merely as the effects of disuse. But this is negatived by the fact that even in complete paralysis of the limbs in consequence of cerebral lesions, very little atrophy occurs, and then only after a long period of time. Friedreich, however, would attribute the atrophy in such cases, as he does in progressive muscular atrophy, to neuritic changes in the terminal filaments of the nerves.

Anstie states "that the periosteum of bone and the fibrous fasciæ in the neighborhood of the painful points of neuralgic nerves not unfrequently take on a condition of subacute inflammation, with marked thickening and tenderness on pressure." It is difficult to determine, however, whether these lesions are primary or secondary. In some cases there is no doubt, from the clinical history of the affection, that the periosteal thickening is the result of simple or rheumatic chronic inflamma-

tion, and has acted as the direct cause of the neuralgia, on account of its pressure upon the nerves.

The trophic disorders of the organs of special sense will be discussed under the heading of trigeminal neuralgia, as they are not observed in any other forms of the disease.

Neuralgia produces various degrees of reaction upon the general system, and it is an interesting fact that these effects are much more marked in affections of the trigeminus than in other varieties, even though the latter equal the former in intensity. A patient may suffer for years from terrible neuralgia of the limbs, while his general condition remains excellent, despite the helplessness to which he may be doomed. In addition, these neuralgias do not often produce a very depressing effect upon the mind, although I once observed a patient who suffered such terrible agony during a first attack of sciatica (and after the paroxysm had only lasted an hour) that it was with great difficulty he was prevented from committing suicide. The ulterior effects of trigeminal neuralgia are much more marked, however, although, even in this form, many patients suffering from severe and intractable pain, present a perfectly healthy appearance. But very frequently these patients are forced to keep themselves on restricted diet, as the least movement of the jaws in deglutition may suffice to develop a severe paroxysm of pain, and they prefer to starve themselves rather than to undergo their terrible tortures. Some of these sufferers are even afraid to speak, dreading a renewal of the attack from this cause. It seems to me that intense and long-continued pain must markedly diminish the nutritive changes occurring in the economy, as I am unable to explain, in any other way, the fact that the loss of weight in these patients is not commensurate with the lessened ingestion of food. Neuralgia also produces, at times, serious effects upon the mind. Apart from the suicidal tendency, which so frequently arises in tic douloureux, melancholia is apt to develop, and the prognosis as regards recovery from the mental disturbance is not very good. It is frequently, however, difficult to determine whether the melancholia is due to the neuralgia, or whether they are both the expressions of a deep-seated disorder in the central nervous system. The family history is sometimes of importance in determining this point.

There is very little to be said with regard to the inter-paroxysmal period of neuralgia, as almost the entire interest centres in the paroxysms. During the intervals pain is entirely absent in mild cases, but in severe ones there may be considerable dull pain and tenderness along the course of the affected nerves, which is heightened in damp weather, although it may stop short of a paroxysm. The cutaneous anæsthesia, to which we referred in describing the paroxysms, persists in severe and chronic cases, during the intervals of the attacks. Many of the trophic changes to which we called attention above, also continue, and may even persist after the primary disease has entirely subsided.

CHAPTER II.

ETIOLOGY.

General Causes.

Heredity.—As in a large number of other functional neuroses, heredity plays a very important part in the etiology of this affection. This fact possesses considerable importance, from a prophylactic, as well as a therapeutic point of view. Neuralgia is classed, in this respect, with epilepsy, hysteria, chorea, insanity, inebriety, etc. This has been most strongly and clearly shown by Anstie, who has collected a considerable number of cases, in which the hereditary interchangeability of the various neuroses to which we have referred is very distinctly manifested. Anstie appears to think, moreover, that phthisis in the parents is capable of giving rise to the development of neuralgia in the offspring, but, from the great prevalence of the former disease, we should be inclined to think that this is merely a coincidence. At all events, a positive statement concerning such a relation of phthisis and neuralgia could only be substantiated by a very large and accurate array of statistics. We find sometimes that neuralgia appears in several children belonging to the same family, although there is nothing in the family history of the ancestors to account for its production. In one case of this kind which came under my notice, the children of one of the parents inherited the neuralgic tendency. A most striking proof of the allied nature of neuralgia and various functional neuroses is its interchangeability with them in the same patient. The following case is a good illustration of the combination of neuralgia with more serious neuroses: Mrs. I. B., æt. 30 years, married; one brother suffered from chorea; all her brothers and sisters are of a nervous temperament. I could get no history of nervous disease in her parents or other relatives. The patient herself suffered from chorea when she was thirteen years old. She began to menstruate at the age of fourteen years, and the menses have been regular ever since. At the age of sixteen, she had an attack of inflammatory rheumatism, and soon afterward was affected with trigeminal neuralgia. The patient married when eighteen years old, and as soon as she became pregnant, noticed pains "like jumping toothache," in the back of the neck, the right shoulder, and the right arm; the right arm was cold, and she could not move it on account of the severe pain produced thereby. During the next six years the patient had trigeminal neuralgia on the right side. Five years ago, she again became pregnant, and suffered from very intense sciatica, which continued until the birth of the child. Three years ago, she became pregnant a third time, and then had an attack of intense lumbago; she began to act strangely during this pregnancy, and became insane when the child was seven weeks old. She was then removed to an asylum, in which she remained for thirteen months (one and a half years ago), at the end of which time she was discharged

cured. She remained well until six weeks ago. The patient is now in the seventh month of pregnancy, and presents a very anæmic appearance. There is no œdema to be detected in any part of the body, and an examination of the urine revealed nothing abnormal. The patient was in good health, since her discharge from the asylum, until one and a half months ago, when she began to complain of weakness, palpitation of the heart, and shortness of breath on exercise. At this time she also began to suffer from melancholy, lost her spirits, disliked company, and thought that "something terrible was going to happen"; she also suffered from sleeplessness during this time. For the last few weeks, she has begun to complain of dull pains, interspersed occasionally with shooting pains in the right shoulder; during the past week she has suffered from right sciatica, which is well marked at the present time, and presents all the characteristic symptoms. She states that she feels just as she did before her previous attack of insanity. The patient is perfectly regular in her habits; does not indulge in stimulants nor excessive sexual intercourse.

I ordered a glass of porter daily, and placed the patient on the tincture of the chloride of iron internally, and administered nitrite of amyl by inhalation (three drops three times a day). The patient began to improve immediately, and within less than three weeks had entirely recovered, the neuralgia as well as the tendency to melancholia having disappeared. Six months later there was a slight return of the former symptoms, but a few days of similar treatment restored the patient to excellent health and spirits.

It is not extremely unfrequent to find that neuralgia, epilepsy, and insanity, appear in the same individual at different periods of life. In fact, Trousseau has described a form of trigeminal neuralgia which he calls *tic epileptiform*, and which he regards as very similar to true epilepsy. We shall refer to this subject again in describing trigeminal neuralgia. It is characteristic of those neuralgias which are due to heredity that they are of a very severe type and intractable to treatment. Anstie has found that heredity influences the development of neuralgia in the most various parts of the body, but for my own part, I have only found its effects distinctly marked in affections of the trigeminus and sciatic.

The predisposition to neuralgia may be acquired not only from heredity but also during the developmental period of youth, from overtaxing of the mental powers. Anstie laid great stress on the importance of this factor, especially when combined with a false, sentimental religious training. But we doubt whether this element is as prevalent now, even in England, as it was ten years ago (when Anstie wrote), thanks to the impetus which has been given of late years, by physicians and educators, to more physiological methods of teaching. In our own country, at least, we know from actual observation that the false, forcing plan of education has been done away with in considerable part. But, although great reforms are being consummated in this direction, nevertheless much still remains to be done, which requires the careful and thoughtful consideration of our profession.

Sex.—It would appear from the experience of most authors that the female sex presents a greater tendency to the development of neuralgic affections than the male sex. Among 178 cases which came under my own observation, 108 were females and 70 males. I have not included in this classification those cases in which the neuralgia formed part of

the symptomatology of hysteria, which is notoriously much more common among females, and an enumeration of which would, therefore, increase the above-mentioned disproportion to a still greater extent. When we come to examine the individual varieties of neuralgia, we find that the female sex predominates in one form, and the male in another.

Thus among ninety-five cases of trigeminal neuralgia, seventy-one were females, and only twenty-four males, while among twenty-five cases of sciatica there were eight females and seventeen males. In intercostal neuralgia the latter relation is again reversed, and the large majority of cases are found in females. In the articles on the special forms of neuralgia we shall enter more in detail into these various considerations.

Age.—An analysis of my cases shows that from the tenth to the twentieth years of life, there were fourteen cases; from the twentieth to the thirtieth years, forty-eight cases; from the thirtieth to the fortieth years, forty-seven cases; from the fortieth to the fiftieth years, thirty-four cases; from the fiftieth to the sixtieth years, nineteen cases; from the sixtieth to the seventieth years, twelve cases; and from the seventieth to the eightieth years, four cases. We therefore find that the first ten years of childhood present no tendency toward the development of neuralgia. This fact is also demonstrated by an analysis of 543 cases reported by Valleix, Eulenburg, and Erb, among which number there were only three cases below the age of ten years.

The largest number occur between the ages of twenty and forty years, and the sexes are equally represented in proportion to the number of neuralgics. Our statistics, therefore, run counter to the general opinion that the largest number of cases of the disease appear in women before the thirtieth year. They also serve to contradict the view that the tendency to neuralgia disappears almost entirely after the age of sixty. From the ages of sixty to eighty years, I observed sixteen cases, which, when compared with the number in other bicennial periods, does not by any means show a diminution in the neuralgic tendency, if we take into consideration the smaller number of individuals at such an advanced age. We should also remember that neuralgias of old age are usually the most intractable to treatment, and they are, in all probability, due to organic changes in the central nervous structures, brought about by the general decay and by the atheromatous changes so frequently observed in the arteries at this period of life.

Weather.—Not only do patients, as a rule, suffer most in damp and windy weather, but primary attacks are also apt to develop under such conditions. This is not true, however, of all varieties of neuralgia. Thus it is not often observed in trigeminal neuralgia, compared with the large number of cases of this variety. In occipital and sciatic neuralgias, however, this is a very frequent mode of causation. I have often observed that sciatica is not uncommon in coach drivers, in whom the constant sitting position and the exposure to inclement weather act together in producing very severe forms of the disease. The manner in which these causes act in producing the disease (as well as all other "colds"), is entirely unknown, and it would be idle and unprofitable to enter into a discussion of the various theories which have been advanced to explain their *modus operandi*, since all are insufficient, and are based on more or less hypothetical grounds.

Sexual system.—Great stress has been laid by some authors upon the influence of the period of puberty, especially in girls, when connected with deficient or painful menstruation, and of the menopause, in producing

various kinds of neuralgias. But we cannot agree with this view. Nervous disorders not infrequently appear at these periods, but they do not, except in comparatively rare cases, assume the neuralgic type.

During the period of puberty hysteria is apt to be developed, and neuralgia not infrequently appears as one of the symptoms of this latter disease. But the affection then presents certain characteristics of its hysterical origin, and should not be classed among true neuralgias. The menopause is also liable to be attended with a peculiar nervous disorder, the chief characteristics of which are sudden flashes of heat, or heat followed by cold, which usually start from the stomach and pass up the chest or to the back ; sudden attacks of perspiration, which arise without any provocation and only last a short time; dizziness; not infrequently, marked increase of sexual desire. This condition is sometimes complicated by a serious depression of spirits, which may pass into melancholia, often tinged with a religious element.[1] But we have very rarely had reason to attribute an attack of neuralgia to this cause; all those which did appear to have any connection with the menopause were cases of trigeminal neuralgia.

We entertain similar views with regard to the effects of excessive sexual intercourse or masturbation. While these factors are liable to cause a condition of nervous exhaustion attended with vague pains in the head and in various other parts of the body, these do not often assume the characteristics which we have described in the chapter on the clinical history of neuralgia.

Depressed general health.—Under this heading we include anæmia, arising from direct loss of blood, excessive lactation, long-continued and exhausting diarrhœa, etc., and the cachexiæ, caused by the development of carcinoma, pulmonary phthisis, etc. When the general health and vitality are lowered by any of these causes, neuralgia finds a fertile field for its production. These causes appear to affect chiefly the trigeminal and intercostal nerves, and the etiological relation between the anæmia and neuralgia is conclusively shown by the disappearance of the latter as soon as the former is removed. In the cachexiæ developing during the course of incurable diseases, the neuralgia produced is apt to continue with increasing severity until death.

Constitutional diseases.—Syphilis may act as a cause of neuralgia in two different ways. In the first place, it may produce pressure upon the nerves in any part of their course, either from thickening of the bones or periosteum, or the development of gummy tumors in adjacent tissues, or from changes in the nerves themselves, such as thickening of the nerve sheaths, hyperplasia of the connective tissue between the nerve-fibres, or gummy infiltration into the nerves. The effect of such lesions is patent, and neuralgias due to them may affect any of the nerves of the body on account of the irregularly disseminated character of these lesions in the tertiary stage of syphilis.

It appears, however, that not only may the disease be produced by direct syphilitic lesions of nerves, but that it may be also due to the direct action of the syphilitic virus. I have neither been able to find any men-

---

[1] We may here remark that the bromide of potassium, in thirty-grain doses t.i.d., acts like a charm in the majority of these patients. Whenever the melancholic tendency is present, we have derived considerable benefit from the inhalation of nitrite of amyl, gtt. iij. t.i.d. After a while this dose may be increased, as the patients are apt to become habituated to it.

tion made of this circumstance in the works on neurology which I have consulted, nor have I met with any cases in practice; but Fournier, the distinguished French syphilographer, states that he has observed cases of supra-orbital and sciatic neuralgia during secondary syphilis, and in which there was no appreciable lesion of the nerves. This view is also advanced by Dr. Keyes in his recent work published in this series. He states that "the essential influence of the syphilitic poison, without physical lesion, doubtless occasions some nervous symptoms, especially early in the disease, such as neuralgias, inordinate appetite, sciatica, local areas of analgesia and anæsthesia at the backs of the hands, and elsewhere." A knowledge of this fact is of course important with regard to treatment.

Blood-poisoning.—Blood-poisoning, due to infection with pus, etc., is not infrequently attended, if the patient recovers from the immediate effects of the disease, by terrible and widely diffused neuralgias which often last for years. The disease is so diffused that we must attribute it to the poisonous action of the material which has been introduced into the blood upon the sensory portions of the nerves or nerve-roots. The distinguished German surgeon, Pitha, was himself a sufferer from excruciating neuralgias due to this cause, and has given an admirable and graphic account of his disease,[1] which is of such an interesting character that we shall give a brief abstract of it.

Case I.—"In the early acute stage, the two shoulder-joints were successively attacked by the most violent boring pains, which, after lasting for hours, suddenly and completely disappeared. Hence the pain darted off to the pelvic region, affecting the bladder, especially its neck, and then the entire urethra. At a later period the pain was confined to the neck of the bladder, simulating perfectly all the symptoms of stone. I felt with the utmost distinctness the spicula at the surface of the calculus being forced, during the paroxysm, into the orifice of the bladder. Gradually I lost this sensation completely, and it only accidentally appeared again in a milder degree at the end of two years. While jumping over a ditch I suddenly experienced the sensation of a stone in the bladder striking against the symphisis. So plainly did the existence of the stone seem on various occasions, that all the preparations were made for lithotripsy, but, to my great astonishment, the most careful exploration of the bladder, repeated five times, failed to discover the calculus, and I became convinced at last that it did not exist. The most severe neuralgia which I at present suffer from affects the heel; and the pain at its greatest severity takes on exactly the form as if the periosteum were being separated from the os calcis. . . . . Omalgia, cystodynia, proctalgia, and neuralgia intercostalis, ulnaris, ischiadica, peronea, cruralis, and digitorum manus et pedis, tortured me one after another, and often several simultaneously, sometimes only for a short time, and at others for days together, producing an amount of suffering that was difficult to endure."

Malaria.—In malarial districts this constitutes one of the most important factors in the etiology of neuralgia. Even in our own city it forms a considerable contingent in the latter affection. In the majority of cases it affects the supra-orbital branch of the trigeminus (popularly known as brow-ague), and the paroxysm of pain appears instead of the

[1] Med. Times and Gazette, 1875, ii., p. 356.

full-blown malarial attack. Other nerves are much more rarely affected, though there is hardly a sensory nerve in the body which may not become involved. Next to the trigeminus, the sciatic nerve is most frequently the seat of pain, and then follow the other nerves without any distinct preference. As we have stated in the chapter on clinical history, even neuralgias which are undoubtedly due to organic affections of the nerves, may run a distinctly periodical course, so that this feature is not pathognomonic of the malarial character of the affection. We are not justified in attributing an attack of neuralgia to malaria, unless we find upon inquiry that the patient has been subject to malarial influences, or can detect an enlargement of the spleen upon physical examination.[1]

Rheumatism, etc.—The loose manner in which this term has been employed in medical literature, has been the cause of a great deal of confusion. The laity frequently use the terms neuralgia and rheumatism interchangeably, and so-called rheumatic influences are regarded as frequent causes of the former affection. But rheumatism, in the strict sense of that term, is very rarely the cause of neuralgia, and, in these rare cases, it appears that the sciatic nerve is always the one involved. In former times gout was also regarded as a frequent cause of neuralgia, but the simple gouty diathesis does not often act in this manner. Whenever neuralgia develops in gouty patients in consequence of the latter process, it is almost always due, as Anstie has pointed out, either to the malnutrition of the nervous system induced by the changes in the blood-vessels, or to the pressure of gouty deposits in the joints, tendons, etc., upon adjacent nerves.

Neuralgias may also develop during convalescence from small-pox, scarlatina, rubeola and typhoid fever, but it is very probable that in these cases the disease is similar to the other nervous disturbances, such as peripheral paralysis, circumscribed atrophy of muscles, etc., which are observed from time to time after these infectious diseases. Nothnagel has also called attention to the development of well-marked neuralgia in the first stages of typhoid fever. This must not be confounded with the cutaneous hyperæsthesia (which is sometimes extremely acute) not uncommonly observed in the first week or two of the disease, and which is entirely distinct from neuralgia.

Lead, mercurial, and arsenic poisoning are also regarded as causes of this disease, but the pains to which these affections give rise, though they are sudden and shooting in character, are situated chiefly in the muscles, and are not confined to the district of a single nerve. We are hardly justified, therefore, in considering them as evidences of true neuralgia.

We have a case under observation at present in which wandering neuralgic pains developed in the lower limbs as a sequel of acute arsenical poisoning. These were soon followed, however, by atrophy and paralysis of the limbs, some loss of sensation, and paralysis of the bladder. The symptoms are probably due to subacute transverse myelitis, and the neuralgic pains were merely a part symptom of this affection.

Alcohol and tobacco.—The excessive use of these substances is also productive of bad results in this direction, not so much as an effect of

---

[1] We wish to call attention to the fact that the opinion laid down in some of the text-books, with regard to the position of the spleen, is erroneous, and that the organ does not normally extend forward beyond the middle of the axillary space. This was first pointed out to me several years ago by my friend Dr. Janeway, and I have *since* then frequently verified his opinion by observations upon the cadaver.

acute intoxication, but rather of their long-continued introduction into the system. The immoderate use of tobacco sometimes gives rise to attacks of angina pectoris, which is undoubtedly a true neuralgia, but a consideration of which is beyond our province at the present time, since we are only discussing neuralgias of the superficial nerves. Aside from this form tobacco is very rarely a cause of the affection.

Alcohol in very rare cases may produce neuralgia as a result of acute intoxication. Thus, I have seen three cases of sciatica due to acute alcoholism. As a rule, however, alcoholism produces these effects as the result of long-continued drinking, in consequence of changes in the blood-vessels, etc. The headaches to which drinkers are so frequently subject cannot be classed, however, under the category of true neuralgias. They are due to cerebral pachymeningitis (which is very often observed in drunkards) to congestion of the brain, to Bright's disease (cirrhotic or "whiskey" kidney), gastric catarrh, etc.

Local Causes.

The term local causes refers to those which act directly upon the nerves, either at their origin in the cerebral or spinal centres, during their course to the periphery, or perhaps even at the peripheral terminations. Within the cranial cavity this category includes tumors at the base of the brain which press directly upon the Gasserian ganglion, periostitis of the petrous portion of the temporal bones, aneurism of the internal carotid. In the spinal canal, it comprises tumors of the cords, localized meningitis, pachymeningitis, and peripachymeningitis, spondylitis deformans, caries and cancer of the vertebræ, the pressure from aneurisms of the aorta.

During the course of the nerves, neuralgia may be produced by pressure from without, such as from aneurismal dilatation of adjacent vessels, enlarged cheesy or calcareous glands, various neoplasms, the pressure of the intestine (herniæ into the sciatic and obdurator foramina), or its contents, pressure of dilated venous plexuses, periosteal thickenings (either of bones across which the nerves pass, or of bony canals which they traverse). Neuralgia may also be due to lesions of the nerves themselves, such as those produced by injury, idiopathic neuritis, or the development of tumors in the nerves (neuromata, pseudo-neuromata, gliomata, etc.). We shall refer to these special causes more in detail when we describe the individual varieties of the disease.

Reflex Causes.

By reflex neuralgias we mean those in which the cause is situated within the distribution of a nerve other than that which is the site of the neuralgic pain. One of the most important factors in this category is functional disturbance of the organs of sight. Dr. George T. Stevens (*Medical Record*, October 13, 1877), has advanced the following propositions in this connection :

1. Among centripetal influences which generate neuralgia, the irritability arising from a perp exity or exhaustion of nerves engaged in the function of accommodation of the eye, must be regarded as by far the most frequent and important.

2. Many inveterate cases of chronic neuralgia not amenable to other forms of treatment, readily yield to the simple process of relieving the eye from irritation resulting from direct accommodation.

These views are confirmatory of the statements of Anstie, who also lays great stress upon the efficacy of eye troubles in the production of neuralgia. But these opinions appear to us to be exaggerated. In our own experience, at least, this cause has only been operative in a small number of cases, producing in the large majority of individuals other symptoms, such as dull headache, inability on the part of the patient to apply himself steadily to any mental work, a general tired feeling, and sometimes considerable dizziness, etc.

Caries of the teeth is a frequent source of neuralgia, especially in the branches of the trigeminus. But Anstie mentions a peculiar and interesting case in which uterine neuralgia was immediately relieved by the removal of a carious tooth. Salter has also called attention to the comparative frequency of cervico-brachial neuralgias which are due to this cause.

Foreign bodies in the various cavities of the head may also give rise to eccentric neuralgias, and, in one instance, a neuralgia of twelve years standing was relieved permanently after the removal of a foreign body from the cheek.

Reflex neuralgias may develop from various functional or organic disorders of the intestinal tract.

The genito-urinary system, both in the male and female, also figure not infrequently among the causes of the disease. This is especially true of uterine disorders, to which are attributed numerous neuralgias, especially those of the lower limbs. The following interesting case, reported by Hunt,[1] is one in which there can be no doubt of the reflex origin of the neuralgic affection. "The patient began to suffer from severe neuralgic pains along the course of the trigeminus during the seventh month of pregnancy. Premature delivery began during the second night after the beginning of the neuralgia; the pain attained its maximum during parturition, but ceased after its completion. When the hand was introduced into the uterus in order to remove the placenta, the pain returned with great severity, and lasted while it was being removed. It vanished immediately after this was done."

Mauriac[2] has called attention to the comparative frequency of reflex neuralgias in various parts of the body, during the course of gonorrhœal orchi-epididymitis.

In addition to cases in which the irritation is situated in the viscera, others are reported in which injury in the course of one nerve has produced neuralgia in the distribution of another. Such cases are extremely rare, and we have only found records of them as occurring in the trigeminus, occipital, and brachial nerves. Thus, an injury to the ulnar nerve has been known to produce trigeminal neuralgia.

---

[1] On the Nature and Treatment of Tic Douloureux, Sciatica, and other Neuralgic Disorders, London, 1844, p. 99.

[2] Gaz. méd. de Paris, Dec., 1878.

CHAPTER III.

PATHOLOGY.

As in all the other functional neuroses, this field of investigation is extremely unsatisfactory. The pathological anatomy of neuralgia has been the subject of laborious investigation, but the results are very meagre. In a considerable proportion of the cases in which it was possible to examine the condition of the affected nerves, either post-mortem or after the operation of neurectomy, no anatomical lesions were discovered. Billroth states that he has so frequently obtained negative results, that "he has become tired of making the examination." At other times various macroscopic and microscopic lesions are found, not alone in the nerve-tissues themselves, but also in the surrounding structures. The following are the lesions which have been observed: congestion and thickening of the sheath of the nerve; sclerotic changes and atrophy of the nerve itself; neuromata, true and false; tumors growing from the surrounding tissues and pressing upon the nerves; simple atrophy of the nerves; capillary hemorrhages into their substance; granular degeneration of the axis cylinders; hypertrophy of some of the fibres, and atrophy of others; inflammation of the ganglia on the roots of the nerves; calcareous degeneration of these ganglia; atrophy from pressure of exostoses, tumors, retracting inflammatory processes, etc., etc.

It is very evident, however, from the negative character of the examination in so many cases, and from the multiplicity of the lesions which have been observed in others, that there are no anatomical characters peculiar to neuralgia. It is even doubtful whether many of these lesions are not of a secondary nature, caused by the often repeated vascular disturbance in the nerves (occurring in the paroxysm of pain), and by the trophic changes so frequent in this disease.

The site of the affection has been variously located by different writers, but the most plausible theory is that advocated with great enthusiasm by Anstie, who believes that the morbid process is situated in the posterior roots of the nerves, or in the gray matter immediately connected with them.

This observer believes that "the morbid change in the nerve-centre is probably, in the vast majority of cases, an interstitial atrophy, tending either to recovery, or to the gradual establishment of gray degeneration, or yellow atrophy, of considerable portions or the whole of the posterior root, and the commencement of the sensory trunk as far as the ganglion." The great objection to his views is that these lesions have never been found in neuralgia, although frequent examinations have been made in the hope of discovering them. But although this is true, Anstie nevertheless adduces weighty reasons for locating the disease in this situation, and it may very well be that his views are correct, in this respect, although the lesion is perhaps of a molecular or chemical nature. Among

the arguments which he has brought forward to show the central nature of neuralgia are:

1. The disease is undoubtedly an hereditary neurosis in numerous instances, and, as such, must be due to some changes in the central nervous system.

2. There are certain organic diseases of the spinal cord, notably locomotor ataxia, in which neuralgic pains play an important part in the symptomatology. In addition, it must be remembered that locomotor ataxia is a disease of the posterior columns of the spinal cord, and that, in some cases, some of the fibres of the posterior roots of the nerves have been found implicated in the sclerotic changes. Even in cases in which the posterior nerve-roots present no anatomical lesions, it is probable that they are in a condition of irritation.

3. The peripheral irritation of a particular sensory nerve may produce neuralgia in nerves which are connected with the irritated one only through the spinal centre.. This is incontrovertible proof that some disturbance must have been transmitted through the sensory portions of the cord, as such a reflex transmission is explicable in no other manner. The secondary implication of the spinal cord after lesions of the peripheral nerves, is admitted also in the theory of neuritis migrans, and Anstie states that, although the lesion of the posterior nerve-roots in neuralgia is usually non-inflammatory in its origin, nevertheless, in rare instances, it may consist of a localized centripetal myelitis, secondary to inflammation of the primarily affected nerve.

4. Unless the neuralgia is very slight and of short duration, it is generally accompanied by vaso-motor spasm, secretory, sensory, motor, and trophic disorders. This combination of symptoms is also most readily explained by a lesion situated in the position claimed by Anstie.

There are some cases, however, to which this theory will not by any means apply. How, for instance, can we accept the doctrine of a central lesion in a case in which a trigeminal neuralgia, which had lasted for twelve years, was immediately and permanently removed by the removal of a foreign body from the cheek?

Then again there are numerous other instances in which neuritis or injuries to nerves near their periphery, etc., give rise to neuralgia, and in these cases likewise it is more plausible to assume a peripheral origin of the neuralgic affection.

But, in spite of all the objections which may be advanced against Anstie's theory, it is the one which is capable of explaining the majority of phenomena observed in the course of neuralgia, especially in those in which heredity is a prominent etiological factor, or in which the affection is due to a constitutional cause.

Uspensky[1] has propounded a theory of neuralgia, based on physiological considerations, which is analogous to that arrived at by Anstie as the result of his profound investigations into the clinical history of the disease. Schiff, Heidenhain, and Ranke had shown that irritation of the nerves produces a rise of temperature and changes the normal alkaline reaction of the nervous tissues to a neutral reaction in the nerves and an acid reaction in the nerve-centres (on account of the formation of lactic acid and the acid phosphate of soda). These products of disassimilation irritate the nervous system, and Uspensky considers himself justified in the opinion "that every pain is either produced, or at least accompanied,

[1] Deutsch. Arch. f. Klin. Med. 1876.

by the formation of a certain quantity of these products of disassimilation in the nerve-tissues, and by their effect upon the nervous system."

He sums up his theory in the following words: "Under the influence of a constant but weak irritation of the peripheral nerves, the sensory nerve-cells of the posterior horns of the spinal cord are continually called into action, and produce, as a reflex effect, a peristaltic contraction of the vessels, which leads to the absorption of the products of nervous energy. In the course of time the absorption diminishes on account of previous increased activity, and the products which irritate the nervous tissues then begin to accumulate. When they have reached a certain amount they first produce, on account of the irritation of the sensory nerve-cells, tetanic contraction of the vessels, and then paralytic dilatation. Repeated recurrence of the circulatory disturbance may lead to atrophy of the sensory nerve-cells, and cause a change in the calibre of the vessels, with loss of their tonus."

While this theory is very ingenious and the conclusions at which Uspensky arrives with regard to the localization of the lesion agree, in the main, with those reached by Anstie, nevertheless his argument is based on purely hypothetical grounds, and some of his premises are more than problematical.

Benedikt, finally, also holds somewhat similar views, maintaining that a considerable number of neuralgias are due to neuritis of the primarily affected nerve, which, spreading upward along the nerves, may finally lead to atrophy of the posterior horns. He also believes that the changes at the bottom of neuralgia may be primarily situated in the spinal cord, and be due to circulatory disturbances in the posterior horns, or to slight meningeal inflammations at the posterior part of the cord. The great objection to this view, as to the others mentioned above, is that, on the one hand, these lesions are not always present in neuralgia, and that, on the other, they may be present, although neuralgia is absent.

We are of the opinion that while Anstie's theory probably holds good with regard to a considerable number of neuralgias, notably those which are due to hereditary influences or to constitutional diseases, nevertheless it is far from being conclusively proven. We can therefore only accept it as a good "working theory."

The causation of the puncta dolorosa is another stumbling-block. The usually accepted theory has been that the painful point is the site of a localized neuritis, or at least of congestion, and that these lesions account for the production of pain. We must remember, however, that even if such a local lesion of the nerves were demonstrated (which it is not), the pain on pressure should be felt at the peripheral distribution of the nerves, in the same manner that a blow upon the "funny bone" produces a sensation of tingling in the little and ring fingers.

According to Sandras, the puncta dolorosa do not depend on the condition of the nerves, but rather on that of the adjacent tissues, which render the spots more capable of being pressed upon. The same objections may be urged against this as against the former view.

According to a very plausible theory, the painful spots are explained by the presence in the affected nerve of the recurrent sensory fibres demonstrated by Arloing and Tripier. It is supposed that these fibres terminate in that portion of the nerve corresponding to the painful spot, and that pressure upon the latter will therefore give rise to pain in the part pressed upon. The only objection to this theory is that proof is still wanting that the recurrent fibres do terminate in the spots referred to.

CHAPTER IV.

DIAGNOSIS AND PROGNOSIS.

ALTHOUGH the term neuralgia has been very loosely applied to the most various kinds of painful affections, nevertheless the disease, although symptomatic of numerous underlying processes, is very clearly defined, and, as a rule, readily recognizable.

As we have seen in the chapter on clinical history, neuralgia presents the following characteristics:

1. The pain is always paroxysmal, at least in the beginning, and exacerbations are manifested even after the disease has lasted for years.

2. The paroxysm usually begins suddenly, the pain is shooting, darting, lancinating, boring, etc., and is referred along the course of the nerves.

3. The paroxysm develops spontaneously, or in consequence of some trifling cause not at all commensurate with the severity of the pain produced.

4. In perhaps the majority of cases, puncta dolorosa are observed in some portion of the course of the nerves.

5. Vaso-motor, secretory, or trophic disorders are noticed in a considerable proportion of cases.

There are, of course, other symptoms which are corroborative, but the first three are essential to the diagnosis of neuralgia.

A considerable number of other painful diseases may be mistaken for neuralgia, and perhaps the most frequent one is myalgia, or so-called muscular rheumatism. This affection is sometimes supposed to be closely related to true rheumatism, but such a view is entirely erroneous. The disease is located, not in the joints or fibrous tissues, as in articular rheumatism, but in the muscular or terminal filaments of the nerves.

As in severe neuralgias, the part affected is kept immovable in order to prevent a fresh attack of pain, but, unlike neuralgia, the parts may be subjected to passive motion without increased suffering. In myalgia, in other words, the pain develops on active contraction of the muscles, and not from passive contraction or mere motion of the parts.

The pain of myalgia, also, is diffused over the entire surface of the affected muscles, and is not confined to the course of any special nerves; it is dull and steady, not sharp and paroxysmal as in neuralgia. Furthermore, myalgia is never accompanied by puncta dolorosa; the whole muscle is tender to pressure, and this is especially marked at its origin and insertion, where its structure becomes tendinous. Finally, a very characteristic difference between the two diseases is found in the different *effects of the* faradic current. True neuralgia is usually rendered worse

by a strong current of faradism, while myalgia is always relieved by it, and sometimes cured as if by magic.[1]

Osteocopic pains of syphilis are also often mistaken for neuralgia. They usually occur in the secondary stage, and instead of being of a lancinating character, and running along the course of the nerves, as in true neuralgia, they are of a boring character, and confined to a circumscribed spot upon the surface of the bones. As a rule, to which there are very few exceptions, they present very marked exacerbations at night. Another differential diagnostic point is furnished by the results of treatment, since mercury usually produces excellent results in osteocopic pains, and would probably only exaggerate the symptoms in cases of neuralgia.

In the condition commonly known as cerebral anæmia (but which is only a form of ordinary, general anæmia), pains in the head are almost always observed, and are very frequently mistaken for neuralgia. They present, however, very marked differences. The pains of anæmia are usually diffused over the entire vertex, are attended with a sensation of heat, and, instead of being paroxysmal, are continuous, dull, and of a peculiar lifting character (they are sometimes likened to a pot of boiling water). The entire scalp may be excessively tender on pressure, but there are no true puncta dolorosa. In addition, the patients usually suffer from insomnia (although they complain of drowsiness during the day), from palpitation of the heart, shortness of breath on exercise, and all the other well-known signs of anæmia. The conjunctivæ will usually be found paler than normal, although the patients not infrequently present a good complexion.

In certain cases neuralgia may be mistaken for neuritis, and, in the minds of a great many, these are convertible terms. As we have repeatedly shown, the most intense neuralgia may develop, although the affected nerve is entirely normal (this is rendered most evident when we remember that neuralgia may develop as the result of an affection of an entirely different part of the body from that which is the seat of the pain), and, on the contrary, neuritis may be well marked without the development of neuralgia. Nevertheless, it is sometimes very difficult, or even impossible, to differentiate these affections, and the two conditions may even be combined. In neuritis, the pain is usually continuous, though it sometimes presents remissions, the entire course of the affected nerve is tender (when superficial, it is sometimes distinctly swollen), and there are no puncta dolorosa; motor paralysis and localized atrophy of muscles soon become evident in severe cases. We must confess, however, that some of these differential symptoms may be absent, and we may therefore be unable to determine the exact nature of the malady with which we have to deal.

As a rule, spinal irritation is readily distinguished from true neuralgia. The most marked differential point is the wandering character of the pains in the former affection. Spinal tenderness is a prominent symptom, and the tender spinous processes correspond somewhat to Trousseau's *points apophysaires*. They differ, however, in the fact that they are apt to vary their location from time to time. Cutaneous hy-

[1] One patient who had very severe myalgia of the back of the neck and shoulders, and who had been thereby incapacitated from work for two months, was entirely cured after one application (ten minutes' duration) of as strong a primary faradic current as he could bear; many similar cases have come under my observation.

peræsthesia and various other hysterical symptoms are combined with the spinal tenderness. In some cases, however, a true neuralgia may lead to the development of spinal irritation, and the two affections are not infrequently combined in young females.

Finally, we will consider the differences between neuralgia and the pains of locomotor ataxia. We must remember that pains may for years constitute almost the sole symptom of tabes dorsalis, and they are, therefore, frequently mistaken in the first stage for ordinary neuralgia, thus leading to grave errors both as regards prognosis and treatment. In ataxia, however, the pains are situated in both lower limbs; they are described as being situated sometimes in the muscles, sometimes along the course of the nerves, they are more irregular in their appearance than true neuralgic pains, they rapidly move from one portion of the limbs to another, and are not accompanied by vaso-motor or trophic disorders. Even in this early stage, the patellar tendon reflex is generally absent. But, in the majority of cases, other characteristic symptoms are also present, such as contraction of the pupils, diplopia, atrophy of the optic disc, the cincture feeling, disturbances of the bladder and of the sexual function, anæsthesia of the lower limbs, notably of the soles of the feet, and inco-ordination of movement.

But not alone is it important to differentiate neuralgia from other painful affections, we must also endeavor to determine whether the peripheral nerves or the central nervous system is involved. A careful investigation of the etiology of the case is often of great importance in arriving at a conclusion in this respect. Thus, for instance, if one of the peripheral nerves has been subjected to a trauma, and a neuralgia develops which is strictly confined to the distribution of such nerve, we are perfectly justified in believing that the lesion which has given rise to the neuralgia is situated at the injured part of the nerve. But it is only in a few cases that we can form an opinion so readily and satisfactorily. In many instances we can only arrive at a probable conclusion after carefully studying the attendant complications.

The distribution of the pain is of importance. If all the branches of a nerve are affected, we are warranted in the belief that the lesion is situated either in the trunk of the nerve or at its central origin. If only one branch of the nerve is involved, and this condition persists during the entire course of the disease, the lesion is probably situated in the affected branch, since it is not probable that a lesion which is situated in the trunk of a nerve, will merely select the fibres going to one branch. But the restriction of the symptoms to a small branch does not positively exclude the central nature of the affection, since the fibres of the nerve diverge at the centres, and we can readily understand that a central lesion may involve one set of fibres or cells, to the exclusion of others.

When motor and trophic disturbances are observed within the distribution of the nerve which is the seat of pain, the lesion is almost undoubtedly situated in the trunk of the nerve. This is not true in all cases, as these complications may be reflex in their character, but in such an event they will not often be strictly confined to the course of the painful nerve.

Neuralgias which form complications of cerebral or spinal diseases are accompanied by the symptoms of the primary affections. Cerebral diseases are accompanied by disorders of the special senses, or of the *other* cranial nerves, paralysis of one or more limbs (usually on one side

of the body), epileptiform convulsions, disturbances of the intellect, etc. Spinal cord diseases are attended by oculo-pupillary disorders (when the lesion is in the upper part of the cord), motor paralysis (frequently of a paraplegic type), atrophy of muscles, increased or diminished reflex excitability, sensory disturbances (usually anæsthesia), interference with the functions of the bladder and rectum, etc.

The prognosis of neuralgia depends entirely upon that of its underlying cause. If due to an organic lesion, the prognosis will depend upon that of the latter affection, whether it is of such a nature as to permit removal by internal medication or by the knife, or whether it is of a central nature and not amenable to treatment.

When due to a constitutional cause, the prognosis again depends upon the character of the primary disease. Thus, a syphilitic neuralgia presents an excellent prognosis, while one occurring as the result of a tuberculous cachexia almost always continues until the death of the patient.

Neuralgias of old age present a gloomy prognosis, although this is not so absolutely unfavorable as Trousseau would have us believe. We may, however, regard it as a general rule that a neuralgia which develops after the age of sixty will continue (usually with increasing severity) until the death of the sufferer.

Those cases which are due to heredity do not present any especially bad prognosis with regard to the individual attacks, but they manifest a greater liability to relapse and to the transformation of the disease into some other neurosis.

The large class of neuralgias which is caused by anæmia, etc., usually furnishes a favorable prognosis, and this improves so much the more, the earlier the patient comes under treatment. After neuralgias have lasted for a long time, secondary changes are apt to develop in the nerves and surrounding tissues, and these react unfavorably upon the course of the disease. As we shall see at a later period, the locality of the pain also possesses considerable influence upon its prospects as regards a speedy or tardy recovery.

CHAPTER V.

TREATMENT.

In treating neuralgias, we should, above all, endeavor to determine the cause of the disease, and, if possible, attempt its removal. Thus, in malarial neuralgias, the exhibition of large doses of quinine will usually cause the disappearance of the neuralgic symptoms. In many chronic cases, however, quinine fails us, and we are then compelled to resort to some form of arsenic. I usually prescribe Fowler's solution in five-drop doses t.i.d., and rapidly increase by addition of a drop to each dose until gastric irritability or œdema of the lower lids begins to develop. The drug is then discontinued for a few days, until the gastric irritation has subsided, after which it is administered in doses slightly smaller than those which sufficed to produce the gastric symptoms. It may now be continued in this manner for a long time without giving rise to any disagreeable effects (it should be given in a little water immediately after each meal, before the patient rises from the table). In addition to the use of Fowler's solution, benefit is also derived in these chronic cases from cool sponge-baths of the entire body, followed by vigorous shampooing, and from the administration of brandy or whiskey.

In syphilitic neuralgias, the treatment varies according to the character of the disease. When the pain is produced without any local lesion in the affected nerve, the greatest benefit is derived from the use of some form of mercury; when the neuralgia is due to pressure upon the nerves from syphilitic periostitis, etc., or to syphilitic changes in the nerves themselves, we must employ iodide of potassium. As we have previously remarked concerning the nervous manifestations of tertiary syphilis, we should only stop at that dose of the iodide which cures the affection. We may begin with ten to fifteen grains t.i.d., and increase this dose as the necessities of the case demand. Relief is usually experienced within a few days. Syphilitic neuralgias are not infrequently the forerunners of more serious affections of the nervous system, and the administration of the iodide should therefore be continued for one or two years after the disappearance of the symptoms.

In the comparatively rare cases of true rheumatic neuralgias excellent results are obtained from the administration of salicylic acid. I generally make use of the following formula:

℞. Acid salicylic..... ℥ ij.
Sodæ bicarb..... ℥ ij.
Glycerinæ,
Aquæ.....āā ℥ ij.
M.

Of this mixture, one tablespoonful is to be taken three times a day, shortly after eating, until slight evidences of intoxication become manifest.

In those cases in which heredity or an acquired neuropathic disposition plays an important part, the tone of the nervous system is lowered, and we must endeavor to improve it by means of rest and generous food.

Whenever possible, prophylactic measures should be adopted, and these refer especially to the education of the patients. Childhood and youth are rarely subject to neuralgia, and a great deal may be done at this time to elevate the tone of the nervous system, and thus prevent the development of neuralgia at a later period. The children should not be allowed to go to school until the ages of seven and eight, and should not be subjected to the "cramming" method of education. From a physical point of view, the plan adopted should be that known as the "hardening" method. The patients should be subjected to daily cold washings or baths, regulated and moderate gymnastics, an abundant and varied diet, and, especially, sufficient sleep. Our invariable advice in these cases is, "Let the patients eat as much as their stomachs will digest, and sleep as long as they can." When the patients arrive at more mature years, they should abstain from mental overwork, and shun alcoholic and sexual excesses. The care of the eyes is also an important element, and their condition should always be investigated when the patient is greatly occupied in reading or writing, fine needlework, or any pursuit in which the eyes are subject to continuous strain.

These measures are important, not alone to prevent the production of neuralgia, but also because such patients are apt, under adverse circumstances, to develop other neuroses.

A large number of cases are due to anæmia, and we must then, of course, resort to ferruginous tonics. The nature of the iron preparation is not very important, but the carbonate of iron and, at the present time, dialyzed iron, are most in vogue.[1] In addition, we must endeavor to ascertain and remove the cause of the anæmia.

When the neuralgia is produced by local causes, or when it is of a peripheral nature, special treatment is required. Thus, when the affection is due to the presence of a foreign body, the pressure of a tumor or cicatrix upon the nerve, etc., surgical interference must be resorted to in order to remove the offending substance.

When no cause can be determined, or when it is of an organic nature and cannot be removed, symptomatic treatment is indicated. Among such measures, the use of morphine as a palliative is probably resorted to most frequently. In some cases it acts not alone as a palliative, but also as a direct curative agent. Not very infrequently, a severe neuralgia will be permanently relieved by the administration of a sufficiently large dose of morphine. It would seem, in such cases, as if the entire relief from pain procured by its administration breaks the "painful habit" of the nerve, and thus allows it to recover its tone. We must be very cautious, however, about beginning the use of morphine in neuralgia, and should not resort to it unless compelled by the severity of the pain. Whenever it does become necessary, the morphine should be administered hypodermically, not only because it acts more quickly when given in this manner, but also because a smaller dose is required to produce a given

[1] We should bear in mind that iron preparations should always be given in very small doses, not alone because only a small quantity is absorbed, but also because the unabsorbed portion quickly interferes with the intestinal functions.

effect. The only objection to this method of administration is that the opium habit develops more readily than when the morphine is given by the mouth. After a while, of course, the dose must be increased; and in chronic, incurable cases, almost incredible quantities of the different forms of opium are taken. It is immaterial whether the injection is made over the course of the painful nerve, or at a distance from it. No definite statements can be made concerning the dose, as this must vary with the severity of the pain and the known peculiarities of the patient.

Atropine is also useful as a palliative, though not so generally applicable as morphine. It may be administered either hypodermically or by the mouth, and I have noticed as a curious fact, with regard to this drug, that it sometimes acts more quickly and powerfully when given by the mouth than when it is introduced hypodermically. The initial dose is from gr. $\frac{1}{96}$—$\frac{1}{64}$, to be repeated as the pain returns, until evidences of its physiological effects (dilated pupils, dry throat, red face and chest, rapid pulse) become apparent, after which the remedy is to be discontinued until these symptoms have subsided. Very many persons are peculiarly susceptible to atropine, and I have seen the above mentioned physiological effects, with the addition of restlessness and a tendency to delirium, following the administration of one one-hundredth of a grain. These phenomena sometimes persist for two or three days (in one case we found them last a week) after the discontinuance of the drug. But its palliative effects cannot be compared with those of morphine, and it should only be used when the patient presents an idiosyncrasy with regard to the latter, or when it fails to produce the desired effect.

We also wish to refer to the sedative action of hypodermic injections of simple water. This plan is not much employed, but we have found it useful in quite a number of cases. In order to relieve pain by this method, we merely inject a hypodermic syringeful of water into the subcutaneous cellular tissue. When this method was first proposed (we forget the name of its inventor) hot water was used, but we have since found that similar effects are produced, whatever the temperature of the water may be. Sometimes the injection of one syringeful of water will relieve the most intense pain within a few moments, at other times the sedative action develops slowly, and several minutes elapse before its effects are produced. In the majority of cases, however, it produces little or no effect, and we are unable to determine beforehand what the result of this manipulation will be. It would be well if systematic investigations were instituted, in order to determine to what cases this method is applicable. Its modus operandi in relieving pain is unknown, but that the opinion which attributes its sedative action to the results of the imagination is erroneous, is proven by the following personal case, which is interesting in very many other particulars:

Case II.—Mrs. A. B., æt. 54 years, married; no hereditary tendency known, but her children present a marked neuropathic disposition (epilepsy, hysteria, nervosisme). Two years ago (1873), Mrs. B. injured her left breast as she was leaving a street car, and soon afterward a small, hard, painless lump appeared at the site of injury; subsequent developments proved this to be a scirrhous growth.

I was first called to see the patient toward the end of April, 1875, and found her suffering from a violent attack of neuralgia, the pain migrating into various parts of the trunk and upper extremities; the attack had developed quite suddenly. After a few days the neuralgic

affection became strictly periodical in its appearance, being attended with severe chills and regularly intermittent variations in temperature of the quotidian type. At the end of a week the attack succumbed to forty-five grain doses of quinine *per diem*. The patient remained well for only three weeks, when the neuralgia recurred, the pains becoming gradually more and more atrocious in character. This attack lasted until the middle of August. None of the many drugs employed produced the slightest beneficial effect except atropia, which, when given to the extent of producing marked dilatation of the pupils, dryness of the pharynx, rapid pulse, and slight tendency to delirium, produced considerable relief. After using it for a few weeks, however, it became entirely inefficient. A peculiar fact noticeable in the employment of the atropine was that, when administered by subcutaneous injection, it produced less effect than when the same amount was given by the mouth. The only relief that could eventually be obtained was from the hypodermic administration of Magendie's solution of morphine, the dose of which was finally pushed to 250 drops *per diem* (often as many as 400) in order to render the patient tolerably comfortable. In conjunction with morphine, I employed subcutaneous injections of water. And here it may be well to mention a very curious fact with regard to the comparative effect on this patient of hypodermic injections of morphine and water. The former always produced a more profound and permanent effect in relieving pain than the latter; but while, in the beginning of the malady, the relief experienced from the morphine was almost instantaneous, and that produced by the water only became palpable after the lapse of four or five minutes, toward the latter stages of the disease this relation was reversed, the relief experienced from the water being instantaneous, while that from the morphine was felt only after the lapse of several minutes.

The neuralgic pains were chiefly confined to the nerves of the upper or lower extremities, but certain of the intercostal nerves were sometimes implicated; at no period during the entire course of the disease did any tenderness on pressure exist over any part of the spinal column. The neuralgia continued, with short intermissions and continually increasing severity, and was only terminated by death (January 31, 1877), the immediate cause of which was acute pleurisy of the left side, complicated with œdema of the lungs.

Autopsy.—The results of the autopsy, which want of time prevented me from making as carefully as was desirable, were substantially as follows:

Brain.—Dura mater: slight pachymeningitis over the convexity of both cerebral hemispheres; a few small cancerous nodules were scattered over the inner surface of dura mater; calvarium normal.

Spinal cord.—Only the dorsal portion of the cord could be examined; the cord itself was entirely free from disease; dura mater: slight pachymeningitis, apparently of about the same date as the cerebral; this membrane was very firmly adherent to the vertebræ, and minute carcinomatous deposits projected from its inner surface.

Vertebræ.—The bodies and laminæ of the dorsal vertebræ (which were the only ones examined) were infiltrated with the cancerous neoplasm to such an extent that they could be cut with the knife; there was no malformation of the vertebræ. The long bones of the body could not be examined.

Mamma.—The tumor of the left breast was nearly as large as a hen's egg, and presented the ordinary appearances of scirrhous cancer.

Lungs.—Right lung slightly œdematous; pleura normal; under the right pleura, and growing from the ribs, were a few small cancerous nodules. Left lung slightly œdematous; left pleura coated with fibrinous exudation; small amount of fluid in pleural cavity.

Liver.—Surface perfectly smooth; size of the organ perhaps a trifle greater than normal; liver-tissue largely infiltrated with carcinoma, the deposits varying in size from that of a pea to a walnut. The consistence of the new growth was a little firmer than that of the normal liver-tissue; the carcinomatous portions occupied at least one-half of the entire bulk of the liver.

Ovaries.—Right ovary was transformed into a scirrhous mass of apparently homogeneous structure, which had attained almost the size of a goose-egg.

The other organs were normal.

Histological appearances.—The new growths in all the organs presented the appearances of carcinoma simplex of a rather small-celled variety; in some places the amount of stroma present was very much diminished, constituting the softer variety of cancer; in other places the relative proportion of stroma to cells became reversed, and the growth presented the appearances of scirrhus.

We will next consider the class of remedies known as counter-irritants, which include the ordinary fly-blister, cantharidal collodion, bisulphide of carbon, electricity, and the actual cautery. I formerly made much more extensive use of counter-irritants in the treatment of neuralgia than I do at the present time, and now restrict their employment almost exclusively to those cases in which there is marked tenderness over the spinous process corresponding to the affected nerve (Trousseau's *point apophysaire*). In such cases counter-irritants usually act admirably; in occipital neuralgia we have found that the application of one or two blisters is generally sufficient to relieve the pain entirely. The use of the ordinary fly-blister and of cantharidal collodion is so common, that it is unnecessary to dwell upon their mode of application.

The bisulphide of carbon is employed by pouring a few drops upon some cotton and then applying this to the skin, the cotton being pressed firmly against the integument in order to prevent evaporation. Within one or two minutes severe pain begins to be felt, and the skin is then found intensely reddened. The cotton should not be applied more than two minutes, as the pain continues to increase for a little while after its removal, and would soon become unendurable. Although the bisulphide produces counter-irritation very rapidly, and is quite ready of application, it possesses the inconveniences of being very painful, and also of being excessively fœtid.

Electricity may also be employed as a counter-irritant. For this purpose we use the strongest secondary faradic current which the patient can bear, the electrode consisting of a wire brush. This is applied over the part to be irritated, the brush being either pressed firmly against the skin or gently stroked to and fro across the surface. The counter-irritation is developed within a few moments, and disappears very soon after the electrode is removed; while the brush is in contact with the skin, however, the pain is very intense, and considerable fortitude is necessary in order to tolerate it for even a few seconds.

The actual cautery with the hot iron is generally included under the head of counter-irritants, but we think that it is erroneously classed among

these agents. Paquelin's cautery may be employed, or an ordinary blast-lamp, using either a glass or an iron rod. When neither of these instruments can be obtained, a poker heated in a good coal fire will suffice for all practical purposes. Most authorities state that the iron should be white-hot, but this is not necessary, and I have always found that a cherry-red heat will answer very well. There is a widespread feeling among the laity (which has been fostered by blood-curdling newspaper articles), that the application of the actual cautery is an heroic measure, entailing excruciating agony. But, on the contrary, its application is almost entirely painless when properly performed. The iron should be applied very gently and rapidly to the skin, so as to leave no scar or suppurating sore, and the entire operation only lasts a fraction of a second. In fact, the pain is sometimes so insignificant, that I have used the iron without the knowledge of the patient, and whenever I could rely upon the statements of my patients in this regard, they have always informed me that the pain of the application was not worth mentioning. It may either be employed on the spine or along the course of the affected nerves. It is a singular fact that the pain may sometimes be relieved by the application of the cautery to a distant part of the body. Thus, there are quite a number of authentic cases on record in which the application of the cautery to the lobe of the ear has relieved an attack of sciatica (this plan was borrowed from veterinary practice). The manner in which the actual cautery produces its beneficial effects is unknown, but there is no doubt that it sometimes relieves neuralgia as if by magic. Even pains which are due to an incurable organic affection, like locomotor ataxia, may be relieved by its agency. It presents this advantage over the ordinary methods of counter-irritation, that it produces no subsequent pain or annoyance, even if it proves unsuccessful as a palliative.

Electricity has also been extensively used as a palliative measure, and both currents have been employed. The faradic current is employed in two ways, viz.: with the wire brush, and with the aid of sponge electrodes placed along the course of the nerves. The former method is really a form of counter-irritation, and we have previously discussed it under that heading. The second method, viz.: that in which sponge electrodes are placed along the course of the affected nerve, is not very much employed at present. My own experience with it has been so unsatisfactory, that I have entirely discarded it for several years past.

The use of galvanism, however, in the treatment of neuralgia is attended with much greater benefit than that derived from the faradic current. It is a much more valuable measure in peripheral neuralgias, and those in which there is no gross anatomical lesion, than in central ones, due to an organic lesion. The majority of authors advocate the use of the descending current. When I first began the systematic use of galvanism in the treatment of neuralgias, I also complied strictly with this rule until I discovered accidentally, while galvanizing the sciatic nerve of a patient suffering from sciatica, that equal relief was experienced when the current was passing in the opposite direction. I have had such frequent opportunity of verifying this experience, that I have become convinced that the direction of the current is entirely immaterial.

The usual method of applying the galvanic current is to place one electrode over the tender point which is present along the spinal column, or, when this is absent, along the course of the nerve, and the other electrode farther down in the course of the nerve near its peripheral distribution. The strength of the current employed should vary according to

the susceptibility of the patient. Beginning with a mild current, its intensity is gradually increased, until a disagreeable burning sensation is produced, and the current is then allowed to flow uninterruptedly for a period varying from five to ten minutes. This should be repeated daily or every other day. We are usually able to determine after the first sitting whether the current will have a beneficial effect or not. In favorable cases a distinct amelioration of the pain is produced lasting from half an hour to one or even two days, and when this palliative effect is marked, the patient is usually cured in from one to three weeks. As a rule, to which there are occasional exceptions, the cases will not be relieved if the cure is not complete within three or four weeks after beginning galvanic treatment. We will find, in the course of our remarks on the special forms of neuralgia, that some varieties are more susceptible to the influence of galvanism than others.

We will next consider the action of those remedies which appear to have a special effect on neuralgic affections.

The number of these so-called specifics is legion, but there are not many which possess any great value. We will only refer to those from which we have derived benefit in practice. These include strychnia, arsenic, phosphorus, gelsemium, aconitia, bromide of potassium, oil of turpentine, nitrite of amyl.

We have only obtained good results from strychnia in trigeminal and sciatic neuralgias, and in the last couple of years have limited its use almost entirely to the treatment of the latter affection. While we do not claim for it a specific action in this disease, we are nevertheless convinced that it relieves more cases than any other single remedy. We should begin its use in doses of one-forty-eighth of a grain given three times a day, and can then increase the quantity administered by one teaspoonful daily until the phosiological effects are produced (stiffness in the lower limbs, and sometimes slight spasms, increased reflex excitability, pain in the throat, nervousness). In bad cases, the patient should be kept slightly under the influence of the drug for two or three weeks, and if recovery does not occur in that period, it will be useless to continue its administration.

The patient should, however, be carefully watched during the entire period of its exhibition, and the drug discontinued as soon as marked effects become evident. Some patients are extremely susceptible to its influence, and I have seen a vigorous adult suffer severely from the administration of three doses of one-forty-eighth of a grain which were taken in the course of twenty-four hours.

Arsenic frequently does good service. It is preferably administered in the form of Fowler's solution (beginning with five drops three times a day and gradually increasing up to ten or twelve drops at a dose).

This drug is one of the best nerve-tonics with which we are acquainted, and may be administered with benefit for a very long time. It is useful, at times, in all forms of neuralgia, but is especially serviceable in those varieties which are apparently combined with degeneration of blood-vessels occurring in old age or in hard drinkers.

Phosphorus, from its presence in the tissues of the brain, was regarded, in former times, as a sine qua non in the treatment of functional nervous diseases. It was, however, gradually losing its prestige in the treatment of neuralgia, when it was again brought strongly to the notice of the profession by Thompson. My own experience has not been very satisfactory with this agent, and I now limit its use to those cases which are accompanied by evidences of nervous depression, especially when caused by

mental overwork. Squibb's solution of phosphorus in cod-liver oil furnishes a very nice method of administration, but I have usually employed it in combination with strychnia and quinine, adopting the formula known as the Hammond mixture.

℞. Strychniæ sulph..... gr. i.
Ferri pyrophosphatis,
Quiniæ sulph.....āā 3 i.
Acid phosphor. dil.,
Syr. zingiberis.....āā ℥ ij.
M. Sig. — 3 i. t.i.d.

Gelsemium sempervirens had been long employed in this country in the treatment of neuralgia, but it did not meet with general favor until the seal of European approbation had been placed upon it. This drug has been chiefly used in trigeminal neuralgia, especially in the dental forms, and my own experience conforms with that of most observers who have used the remedy, viz.: that it relieves some cases with great rapidity, while others of an apparently similar nature are not benefited in the least. The fluid extract is the most reliable preparation, and may be given in doses of gtt. v. to x., t.i.d. The patient should be carefully watched while the drug is being administered, as it has a very powerful paralyzing action upon the heart, and may very rapidly produce great muscular prostration.

Aconitia had been recommended in neuralgia by Benj. Brodie and by Romberg, but afterward fell into disuse until extolled in the highest terms by Gubler several years ago. In this country, Dr. Seguin was prominent in bringing it into public notice, and for a year or more, I have used it extensively. I must confess, however, that my expectations with regard to the effects of this drug have not been entirely realized. While it produced decided relief in the larger number of cases in which it was used, it produced a complete cure in only a few. When the preparation is good (Duquesnel's aconitia[1] is the only reliable article), it is an extremely powerful poison (only equalled in virulence, perhaps, by nicotine) and should be given in exceedingly small doses.

The dose is from $\frac{1}{150}$ — $\frac{1}{125}$ of a grain, given three times a day, and this may be increased by one dose daily until the physiological effects (dryness of the throat, slowness of the pulse, and tingling of the tongue, roof of the mouth, and tips of the fingers) are produced. If immediate relief is not obtained, it should be continued for several weeks before being discarded. Although this remedy does not fulfil the enthusiastic encomiums of Prof. Gubler, it will, I think, be found to be one of the most generally useful of all anti-neuralgics, and in some even desperate cases the pain is found to disappear as soon as the patient is fully under its influence. I also wish to emphasize the fact that, whenever necessary in the treatment of neuralgias, the remedies employed should be pushed to the production of their full physiological effects, before they are renounced as useless. The physician, although avoiding recklessness, should be bold in the use of his medicinal armamentarium.

[1] Mr. Rice, chemist to Bellevue Hospital, informs me by verbal communication, that Duquesnel's aconitia is composed of aconitia proper and of pseudo-aconitia, the effects of the latter being directly antagonistic to those of the former. An English preparation of aconitia, which, according to Mr. Rice, is chemically pure, will soon appear in the market.

Bromide of potassium was, at one time, regarded as a panacea for a considerable number of nervous disorders, and neuralgia was also included in this category. But it has no direct palliative effect whatever in the latter disease. It is useful in those cases which are complicated with hysteria, or with an irritable condition of the nervous system arising from any source, but in such instances it merely soothes the nervous system, and does not relieve neuralgic pain. It must, therefore, always be combined with some other remedy, and large doses are usually required. It is given to advantage with hydrate of chloral in those cases in which the patients suffer from insomnia, when this condition is due to causes other than pain.

Oil of turpentine, though very little employed at the present time, sometimes proves very useful in chronic cases of sciatica. It should be given in doses of half an ounce to an ounce, immediately after meals.

Nitrite of amyl has been recommended of late in the affection under discussion, and quite a number of cases have been reported, in which it has produced happy results. My own experience with it in this disease has been small, and while my results have not been very striking, they are, however, sufficiently satisfactory to stimulate to further trial. The nitrite of amyl should be administered by inhalations, beginning with three-drop doses three times a day, and gradually increasing, as the necessities of the case demand.

Finally, we must devote a little attention to the surgical treatment of neuralgia, although this should be included, strictly speaking, in the province of the practical surgeon. These measures include neurotomy (nerve section), neurectomy (excision of a piece of a nerve), nerve stretching, and ligature of arteries.

Although it has been very conclusively shown that cut nerves do not unite by first intention, nevertheless the union occurs very rapidly, and there is some reason to believe that the regeneration of a piece two inches in length will not occupy a longer time than that of a portion only a fraction of an inch. For this reason, therefore, simple neurotomies should not be resorted to whenever neurectomy can be performed, and, in addition, as large a piece of the nerve as possible should be removed. In the operation of stretching the nerve an incision is made along the length of the nerve, and the latter is laid bare and detached from surrounding tissues; the finger is then introduced beneath the nerve, and vigorous traction made. When this operation is performed upon small nerves, the traction must be exercised with great caution in order to obviate their rupture. This accident happened to Czerny while stretching one of the branches of the trigeminus, and he was compelled, in consequence, to exsect a portion of the torn nerve. The *modus operandi* of the relief obtained by the operation is entirely unknown.

Ligature of the carotid has been employed several times in hopeless cases of tic douloureux, but this operation should only be resorted to as a *dernier ressort.*

Surgical interference is especially indicated when the disease is of a peripheral nature. This does not, however, constitute an absolute rule, as neurectomy has been known to produce a cure when the neuralgia was the result of a central affection. Such an effect is regarded as the result of an "alterative" action upon the nutrition of the central nervous system—another method of expressing our ignorance. Neurectomy is very rarely dangerous, but it should, nevertheless, be only used as a last resort. *The prognosis* of the operation, as regards complete and permanent re-

covery, is not very good. A relapse may occur long after the operation, and Gussenbauer reports one case in which the disease returned five years after exsection.

Five years ago Arloing and Tripier demonstrated that recurrent nerves pass from the peripheral ramifications of one nerve to those of adjacent ones, and therefore pain which is located in the distribution of one nerve may be attributed to an affection of another, and perhaps entirely healthy one. These facts are of great importance, not alone from a physiological, but also from a practical standpoint. They teach us that great caution must be exercised in the determination of the nerve to be operated upon, as it has been found, on more than one occasion, that a portion of the wrong nerve has been exsected, and a second operation therefore rendered necessary. We will discuss this subject more in detail in our remarks on the special forms of neuralgia.

CHAPTER VI.

TRIGEMINAL NEURALGIA.[1]

(Prosopalgia.)

Clinical History.

Trigeminal neuralgia is unilateral in almost all cases, and does not often affect all the branches of the nerve. Whenever this is the case, the pain often radiates into the occipital nerve or cervico-brachial plexus. The supraorbital branch is by far the most frequently involved; and, when this occurs in a malarial patient, the affection is popularly known as brow-ague. Any nerve twig may, however, be separately implicated to the exclusion of all the others, and there are even a few cases on record in which the branch going to the tongue was alone affected.

The paroxysm of pain may develop with extreme rapidity, but there are usually some prodomata for a few hours before the onset. These consist of a feeling of "drawing" in the distribution of the nerve, of numbness, slight wandering pains, or a sensation of coldness. Then slight "stitches" begin to run along the nerve; they soon increase in severity, and appear to dart with fearful rapidity (sometimes running toward the centre as well as toward the periphery). A lull then occurs, during which merely a numb pain is appreciable; but this calm is only temporary, and is interrupted in a moment or two by another attack of pain. The entire paroxysm lasts for a period varying from a few minutes to several hours. Trousseau has described one variety under the title epileptiform neuralgia, which he thinks is analogous to, and, in some instances, is an expression of true epilepsy. This form develops at an advanced age, the pain is extremely intense and always darting in character, and is accompanied by convulsive movements of the side of the face involved (tic douloureux). The patients endeavor to mitigate the pain to a certain extent by firmly compressing the cheek with the hand, and this may be done so forcibly and continuously that the skin is par-

---

[1] The trigeminus is divided into three branches, viz., the ophthalmic, superior maxillary, and inferior maxillary. The *ophthalmic* branch supplies the lachrymal gland, the conjunctiva and integument of the upper lid, the skin of the frontal region as far as the vertex, the mucous membrane of the frontal sinus, the ciliary muscle and iris, the integument of the nose, inner surface of the lower lid, the lachrymal sac, and caruncula. The *superior maxillary* branch supplies the integument of the temple and side of the forehead, the upper teeth, antrum, lower eyelid, side of the nose, the integument and mucous membrane of the upper lip. The *inferior maxillary* branch supplies the integument of the temporal region, the auditory meatus and integument of the ear, the temporo-maxillary articulation, the parotid gland, the mucous membrane of the tongue, mouth, and gums, the lower teeth, integument of the chin and lower part of the face, and the lower lip.

tially rubbed off and the face denuded of the beard; they are unable to take solid food for fear of producing a paroxysm, and for the same reason articulation is interfered with, since even the movements of the cheek may give rise to the pain. When the paroxysm reaches its height, the patients may become delirious for a few moments on account of the atrocious character of the pains. We cannot agree with Trousseau's view of the close relationship of this form of the disease with epilepsy. To our mind it is merely a severe form of neuralgia, occurring at that period of life when arterial degeneration is going on, and the nutrition of the nerves suffers severely in consequence. Nor do we agree with Trousseau in giving such an extremely unfavorable prognosis in this variety. These cases are, at times, combined with melancholia, and this is not surprising if we reflect upon the intensity of the sufferings of the patients and upon the unfavorable prospects as regards recovery which is usually offered to them by physicians.

Puncta dolorosa are very commonly observed, and are readily recognizable. The following ones are observed in neuralgia of the ophthalmic branch: a *supra-orbital* point, at the supra-orbital notch or foramen, a *parietal* point, at the summit of the parietal protuberance, a *nasal* point, at the upper part of the lateral aspect of the nose; in neuralgia of the superior maxillary branch, we find an *infra-orbital* point, at the infra-orbital foramen, a *nasal* point, at the lower part of the lateral aspect of the nose, a *malar* point, over the middle of the malar bone, a *superior gengival* point, in the upper gums; in neuralgia of the inferior maxillary nerve, a *temporal* point, immediately in front of the lobe of the ear, a *mental* point, at the mental foramen, and an *inferior gengival* point, in the lower gums. Other puncta dolorosa have been mentioned by various writers, but these are the only ones which we have been able to detect. We must also refer to the fact that Trousseau's *point apophysaire* is frequently observed over the first or second cervical vertebræ, though it is not by any means so constant as the distinguished French observer believed. Pressure upon the tender spinous process sometimes sends a thrill of pain through the supraorbital nerve.

When the ophthalmic branch is the seat of the disease, numerous concomitant symptoms may be manifested. The most frequent are intense redness of the conjunctiva and profuse secretion of tears; there is not infrequently an erysipelatoid condition of the integument of the forehead. The hair of the eyebrow and of the scalp, in the distribution of the affected nerve may turn gray or become brittle or coarse. Herpes as a complication of trifacial neuralgia is almost exclusively limited to the distribution of the ophthalmic nerve (herpes ophthalmicus). It is usually confined to the upper lid or forehead, but it sometimes involves the cornea, leading to opacity of that organ, which disappears after a time in most cases. It is also liable to produce iritis in such instances from an extension of the corneal inflammation to the subjacent tissues or from the presence of the eruption upon the iris. Quite a number of cases have been reported which appeared to indicate the direct dependence of glaucoma upon neuralgia of the trigeminus. Irritation of the fifth nerve has been known to produce increased tension of the eyeball. But this question appears to be still unsettled, and it remains for further investigations by ophthalmalogists to decide this mooted point. In one interesting and rare variety of ophthalmic neuralgia, the pain is strictly confined to the interior of the eyeball, and is of a tensive boring character. This is usually looked upon as a neuralgia of the ciliary nerves;

sooner or later it is generally combined with neuralgia of some other branch of the trigeminus.

In neuralgia of the superior and inferior maxillary branches, the vaso-motor complications are most marked. The face is red and hot, and perspires profusely, and the carotid throbs violently. The secretion from the nose on the affected side is usually increased, and in one case, I saw it assume a sero-purulent character during the continuance of the pain. The tongue may be furred on the painful side, and the buccal secretions increased in amount. The skin of the cheek is sometimes thinned and shining in chronic cases; it is in this region, also, that I observed the hypertrophy of the integument and subcutaneous cellular tissue in the case mentioned in the general remarks on neuralgia.

Gross has described a peculiar form of neuralgia affecting these two branches in old, toothless people. He attributes it to the compression of the terminal filaments of the dental nerves by the deposit of newly formed osseous tissue in the cavities of the alveolar processes.

Reflex amaurosis has also been attributed to neuralgia of the dental nerves, but this statement needs verification before being fully accepted.

The duration of trigeminal neuralgia frequently depends upon our ability to remove its cause. When the neuralgia develops after the age of fifty, or in consequence of irremediable organic changes, the patients are very liable to suffer from ever-increasing pain until death puts an end to their tortures. The patients (even those suffering from the severer forms) may retain an appearance of health for a long time, but sooner or later, the continuous depressing influence of the pain and the insufficient nutrition of the body caused by their fear of taking food, lead to progressive emaciation, and sometimes to a markedly hysterical frame of mind. Such patients lose their power of will, their judgment becomes impaired, and the emotional nature is unduly excited. A reference to their ailment is sometimes sufficient to cause them to weep profusely. They become unable to attend to business, not so much on account of the pain (some patients are able to go through their usual occupations while suffering terrible tortures, and without manifesting their affliction to those around them), but rather from peevishness and inability to direct their minds to any subject requiring careful thought and attention. Blandford has observed one form of neuralgia which alternates with attacks of insanity.

Etiology.

Sex.—In my own experience, the female sex has suffered twice as often as the male; among 107 patients, 71 were females and 36 males. We should also take into consideration that I have not included, in this number, any cases of hysterical clavus or of migraine, which are regarded by some writers as neuralgic in character, and which are almost exclusively met with in females. This preponderance on the part of the female sex is probably due in great measure to the influence of the child-bearing period, and of the menopause.

Age.—More than half of my cases developed between the ages of twenty to forty years, or that period at which the struggle for life is most severe, and in which the nervous system is accordingly subjected to the greatest amount of wear and tear, in which the males are engrossed in the cares of business, and are pressed by their competitors in the race for a subsistence, and the females are compelled to devote themselves to

the claims of society, or, in the poorer classes, to the earning of their daily bread, in addition to the manifold duties of maternity.

Heredity.—Though I have, unfortunately, no statistics in this respect, my experience has been that an hereditary influence is exercised more frequently in the development of trigeminal than of any other form of neuralgia. This is, no doubt, due to the fact that the brain (which contains the nucleus of origin of the fifth nerve) is more frequently the site of disturbance on account of bad hereditary influences than the spinal cord, from which the sensory nerves of the body originate. An acquired neuropathic disposition may, of course, also lead to the development of trigeminal neuralgia, but we have discussed this subject with sufficient fulness in our general remarks on etiology.

Anæmia, etc.—Trigeminal neuralgia develops, in numerous individuals, as soon as the general condition is somewhat below par, whatever the cause of this deterioration of health may be. Thus, it may be caused by anæmia from direct loss of blood, profuse discharges, etc., by cachexiæ of various kinds (tuberculosis, carcinoma, etc.), or by overwork. Mental overwork is especially effective in this respect, and is much more likely to produce neuralgia when the patient is continually worrying or fretting. One of my friends, who suffers occasionally from slight neuralgic attacks due to this cause, finds that the pain disappears if he takes a hearty meal. Sexual excesses are also injurious, and very frequently act as an exciting cause.

Reflex causes.—A source of irritation situated in the most remote parts of the body may act as the exciting cause of this form of neuralgia. One of the most important causes in this category is straining the eyes, though, as we have previously stated, we are of the opinion that the frequency of this factor is overestimated, because a clear distinction is not usually made between trigeminal neuralgia and other forms of headache. Functional abuse of the eyes usually leads to supraorbital or to ciliary neuralgias.

Disorders of the genital organs and of the intestinal tract may also be included among these causes. In one case I found that a severe attack of trigeminal and occipital neuralgia was caused by the presence of a tapeworm in the intestines, and that the pain disappeared after the expulsion of the latter. But cases of this nature are so infrequent that we are compelled to admit the pre-existence of a neuropathic disposition in such individuals.

Organic lesions.—In the cranial cavity there are several conditions which may act as causes of trigeminal neuralgia. Romberg reported the well-known case in which the autopsy showed that an aneurism of the internal carotid had pressed upon the Gasserian ganglion and the origin of the trigeminus, and had thus given rise to intense neuralgia of many years' duration. Other tumors at the base of the brain (carcinoma, syphiloma, cholesteotoma, etc.), may act in a similar manner. Exostoses growing from the petrous portion of the temporal bone and periostitis of this part should be included in the same category. In the peripheral course of the nerves, they may be irritated by tumors growing from adjacent parts of the face, by wounds of various kinds, the presence of foreign bodies, the spread of inflammation from middle ear troubles, dental caries or exostoses of the alveolar processes.

Constitutional causes.—Malaria and syphilis are the only general diseases which have an undoubted influence upon the production of this form of neuralgia. Malaria is a very important factor, and usually gives rise

to supra-orbital neuralgia, though in rare cases it may produce pain in the distribution of all the branches of the trigeminus. This variety is easily detected by its periodicity and ready amenability to treatment by quinine, or, in chronic cases, by arsenic.

Syphilis may give rise to neuralgia either during the secondary or tertiary stage. The former variety is extremely rare, and no cases have come under my own notice. The latter form is more common, and is due to gummatous infiltration of the nerves or their neurilemma, or to compression of the nerves by gummy growths. But even this latter form is not so frequent as is generally supposed, and the mistake undoubtedly arises from the fact that osteocopic pains are often regarded as neuralgic in character.

Cold.—Sudden checking of perspiration from exposure to a draught, etc., has been regarded as a frequent cause of trigeminal neuralgia, though the *modus operandi* of its action is very obscure. There is no doubt, however, that it sometimes acts as an exciting cause ; we also find in cases of this kind that considerable relief is often obtained by the application of warmth and diaphoresis.

A considerable contingent of cases remains, however, in which no cause is ascertainable, in which we must remain satisfied with the bare diagnosis of neuralgia, and in which merely symptomatic treatment is therefore admissible.

Diagnosis and Prognosis.

The *headache of anæmia* is often mistaken for neuralgia, but it can be readily differentiated. The pain of anæmia is not confined to the course of the nerves, but is diffused over the forehead or the entire vertex. It is of a dull or lifting tensive character, and is continuous, never paroxysmal. The temperature of the scalp is sometimes raised very appreciably, and the integument may be acutely hyperæsthetic.

Migraine (hemicrania) has been regarded by many authorities as a form of neuralgia, but it is now almost universally considered to be a neurosis of the sympathetic nerve. The differential diagnosis between migraine and trigeminal neuralgia is usually effected with great readiness. While neuralgia is rarely observed before the age of twenty, migraine generally begins about the period of puberty, and sometimes even much earlier. Furthermore, heredity plays a much more important part in the etiology of migraine than it does in that of neuralgia. The pain also presents entirely different characteristics. In migraine it is of a throbbing, dull character, and, unlike that of neuralgia, is felt deep within the skull, and not in the distribution of special nerves. Migraine is also sometimes accompanied by hyperæthesia of the auditory and optic nerves, and also by hallucinations of these two senses. The course pursued by the two affections is entirely different. An attack of migraine rarely lasts more than twenty-four hours, and the patient is then free from suffering until another attack occurs. We must not forget, however, that the two affections may be combined in the same patient.

Clavus hystericus.—This is a fixed pain, situated in the parietal region near the sagittal suture ; the suffering has been likened to that produced by driving a nail into the scalp. It is always combined with other well-known symptoms of hysteria. These characteristics are sufficient to *differentiate it from* trigeminal neuralgia.

Osteocopic pains.—These are also frequently mistaken for true neuralgia, when they occur in the forehead. They usually make their appearance during the secondary stage of syphilis, and almost always occur at night; they are confined to one spot, and are sometimes excruciating, "as if the bones were being split." They readily recover under the use of mercurials.

Headache of Bright's disease.—Although the diagnosis between this condition and trigeminal neuralgia is apparently very easy, nevertheless mistakes sometimes occur. Within the last six months, two grave errors of this kind have come under my notice. In one case the patient had been treated for upward of two years for neuralgia. The character of the pain alone should have led to a suspicion of serious organic trouble; it was bilateral, of a dull, continuous character (lasting day and night), and attended with frequent attacks of vomiting. An examination of the patient's heart revealed the existence of hypertrophy of the left ventricle without valvular lesion. Suspicion was therefore directed to the kidneys, and an examination of the urine showed a low specific gravity, and the presence of a very large amount of albumen. The diagnosis of Bright's disease was therefore evident.

The various conditions which we have enumerated above are frequently mistaken for neuralgia. Such an error is chiefly attributable to the loose manner in which the term neuralgia is employed by the profession. If we bear in mind the distinguishing characteristics of neuralgic pain to which we have referred in the general remarks on diagnosis, the differentiation from other painful affections will be readily made in almost all cases.

Prognosis.—This depends in great part upon the etiology. If the neuralgia is due to malaria, syphilis, exposure, anæmia, or overwork, the attack will soon subside, as a rule, when we are in a position to successfully combat the primary cause. The prognosis is also very favorable when the neuralgia is reflex in its nature. A great many of the so-called idiopathic cases, however, especially those which begin late in life, and those which are due to anatomical lesions of the nerves, are very obstinate in their nature, resisting all medical treatment, and sometimes even surgical interference (neurotomy, etc.). A large proportion, also, of those cases which are due to hereditary influence, present a gloomy prognosis as regards complete recovery. Although such an attack of neuralgia may, perhaps, yield readily to treatment, nevertheless there is great liability to relapse, whenever the tone of the nervous system is lowered from any cause, such as mental overwork, sexual or alcoholic excesses, etc. But, on the whole, trigeminal neuralgia is not such a bugbear as it is generally regarded, and there are numerous measures at our command, which, if they will not cure, will at least produce decided relief in most cases, and there are very few in which life cannot be made tolerable to the sufferers.

Treatment.

The remarks which have been made on page 112 *et seq.* will also apply to the treatment of trigeminal neuralgia, but there are some special points to which we desire to call attention. In the first place, severe cases demand entire mental rest, especially when the patient has a bad family history. If there are any evidences of serious hereditary neuroses, especially if there has been any insanity in the family, we must regard

the patient with suspicion. If he is engrossed in the cares and anxieties of business, he should, for a time, give up his occupation when this plan is practicable. Physical rest should also be obtained. The exercise allowed the patient must at first be of a mild character (walking, carriage-riding, fishing, etc.). He must be especially warned against the dangers of sexual excesses, and it is perhaps advisable to counsel absolute continence until the patient has entirely recovered from the neuralgia.

The diet should be varied and plentiful, and the appetite stimulated in every possible way. Unfortunately the movements of mastication will frequently give rise to the development of a paroxysm of pain, and the nutrition of the patient is thus seriously interfered with. We must then place our chief reliance on a milk diet. In those unfortunate cases in which the pain produced during eating is so excruciating that the patients will only take very minute quantities of food at a time, we should, without much delay, begin to nourish the patient by means of rectal alimentation. This is especially important because the severe cases to which we now refer usually occur in old age, and the vital powers are therefore in danger of sinking from the combined depressing influences of the terrible pains and the insufficient amount of nourishment.

In protracted cases the condition of the eyes should always be carefully examined, and if it is found necessary, reading (especially newspapers) should be entirely interdicted.

Other reflex sources of irritation must be carefully inquired into. The condition of the genital organs and of the intestinal tract should also be investigated. We must, however, advise caution in respect to the habit of indiscriminately pulling out carious teeth (sometimes even sound ones) when a patient suffers from a severe attack of neuralgia. In bad cases this is sometimes carried so far that all the teeth are removed from one side of the jaw, the patient, in the meanwhile, growing steadily worse. It is to be remembered that the teeth are essential factors in the process of digestion, and that they should, therefore, not be sacrificed unwarrantably. They should not be extracted until we have made a careful search for other causes, and unless manipulation of the supposed offending tooth produces a decided increase of pain.

In that form of neuralgia known as neuralgia of the jaw-bones, which was first described by Gross (and which he attributed "to the compression of the minute nerves distributed through the wasted alveolar processes, dependent on the encroachment of osseous matter upon the walls of the canals in which they are enclosed"), this distinguished surgeon obtained admirable results by resecting the affected part of the alveolar process. A succinct account of the operation is given in the *American Journ. of Med. Sciences*, for 1870.

Trigeminal neuralgia, due to malaria, must be treated, of course, like all other manifestations of this disease, with large doses of quinine. The best plan is to slightly cinchonize the patient for two or three days, and then continue the remedy for a couple of weeks in tonic doses. This variety presents a very great tendency to relapse whenever the system is subjected to malarial influences.

The syphilitic forms of this neuralgia are treated with mercurials and iodide of potassium respectively, according as they appear in the secondary or tertiary stages. We must repeat, even at the risk of appearing *tiresome*, that these remedies must not be discontinued as soon as the

neuralgia has disappeared, but should be administered for a long time afterward.

Syphilitic neuralgias are very frequently the forerunners of more serious nervous affections, which can, in such cases, only be forestalled by persevering, long-continued anti-syphilitic medication. We should also warn these patients against the dangers of alcoholic excesses. One of the severest forms of cerebral syphilis is that due to changes in the coats of the blood-vessels, and these would only be aggravated by excessive indulgence in stimulants.

In other forms of trigeminal neuralgia resort is had to the use of narcotics or so-called specifics.

In my own practice I have learned to dispense with narcotics to relieve trigeminal neuralgia, until other remedies have failed me. It is to be remembered that this form of neuralgia is especially apt to be long-continued, that in such cases the opium habit is very liable to be established (especially because we must employ the hypodermic method of administration), and that when this has once developed, an attempt to diminish the quantity of morphine injected will almost inevitably lead to a decided increase in the intensity of the pain. This is not an imaginary evil, as the majority, perhaps, of the cases of chronic opium-eating which have fallen under my notice, have been due to the administration of opium begun in this very disease.

But when we have determined to use opium (the best method of administration is the hypodermic injection of Magendie's solution) we should always bear in mind that our object is to relieve the pain as rapidly as possible. It is much better to give a single large dose than a couple of smaller, insufficient ones; by the latter method an equally large amount of opium may enter the system without producing the desired effect. It is impossible to state in so many words what dose is requisite, since this depends entirely upon the severity of the pain, and the individual susceptibility of the patient. In exceptional cases, opium appears to have not only a palliative, but also a markedly curative effect. It is sometimes found that the patient remains permanently free from the pain, as soon as the latter has been entirely relieved through the use of a full dose of the drug. In those cases (and unfortunately they are very frequent) in which narcotics must be employed for a long time, the use of morphine may be alternated from time to time with hypodermics of atropine or of pure water, whenever either of these agents is found serviceable. In this manner we can avoid accustoming the system to the administration of morphine, and can thus restrict the quantity exhibited to the lowest possible amount. Morphine or atropine should never be injected into the face, because they produce no better effect when administered in this manner than when injected at a distance, and furthermore, abscesses develop at the site of injection in exceptional cases, and might thus lead to deformity. Bartholow also recommends aquapuncture for its palliative effects, thirty to sixty drops being injected, and the operation repeated in two minutes if not successful.

In aconitia we possess a remedy which appears to single out trigeminal neuralgia for the display of its curative properties. The initial dose of this remedy is gr. $\frac{1}{120}$, t.i.d., and increased by a single dose daily until its physiological effects are produced. The drug is now prepared in the form of granules, but we do not favor its exhibition in this shape, because we cannot graduate the dose as carefully as is desirable in dealing with such a virulent poison. This remedy has produced recovery in a case

in which all three branches of the trigeminus had been unsuccessfully excised, and I have also seen a patient in whom it produced relief after an unsuccessful excision of the inferior dental nerve had been made. In a certain proportion of cases, however, it fails us entirely, whereas it may produce a perfect success in other and apparently similar ones. But aconitia has already proved a very valuable addition to our anti-neuralgic remedies, and after the enthusiam of some observers has been moderated, and the indications with regard to its use have been more clearly defined, it will assume its position as, perhaps, one of our best remedies for the disease under consideration.

Arsenic, in the form of Fowler's solution, is also a valuable agent. We have previously referred (page 118) to the manner in which it should be administered. This drug appears to be especially indicated in the severe and obstinate cases developing in old age, and we have sometimes been surprised to see what marked and rapid improvement will occur in some which were apparently hopeless. Arsenic does not appear to me to act directly upon the neuralgic affection, but rather by giving tone to the general nervous system. I have arrived at such a conclusion from the fact that it is an invaluable agent in all neuroses due to nervous exhaustion or in which the nutrition of the nervous system is impaired in old age.

Gelsemium sempervirens is especially reliable in dental neuralgias, but is also useful in a considerable number of other varieties of facial neuralgia. This should also be given until slight toxic effects become noticeable, the initial dose varying from five to ten drops of the fluid extract. It is often difficult to obtain a reliable preparation.

Strychnia has also been employed in this affection, but I cannot recommend it very highly, and, at present, I only use it after having made an unsuccessful trial of the drugs previously mentioned. My experience with it has been so unsatisfactory that I am gradually abandoning its use in this disease.

Whenever the affection is attended with tenderness of the first or second cervical vertebræ (*point apophysaire*) it is advisable to employ counter-irritation over the tender spot (either with a fly-blister or with the electrical wire brush) in combination with some of the remedies which we have just mentioned. Counter-irritation, under these conditions, almost always produces a certain amount of relief.

The employment of the faradic current is not only devoid of advantage in this affection, but, on the contrary, I have seen a single application of this form of electricity convert a mild case into an extremely severe one. The galvanic current is not open to this criticism, but its use is attended with fewer successes in this than in any other form of neuralgia. In applying it, one electrode should be placed upon the nape of the neck, and the other over the exit of the branches of the nerve from their bony canals under the integument of the face. The current should only be employed continuously, and, in cases in which we suspect a central origin, we may place both electrodes over the mastoid processes, and allow a current to pass through the brain. As soon as the patient becomes dizzy the application is discontinued, and a weaker current employed. We must be careful to use only mild currents upon the face, and their intensity should be increased very gradually and cautiously. The applications may last from five to ten minutes, and are repeated daily or every other day.

When internal medication or the use of galvanism, etc., prove useless,

we are compelled to fall back upon surgical interference as a *dernier ressort*. In no other form of neuralgia has this mode of treatment been more frequently or more successfully employed. Recourse may be had to neurotomy, neurectomy, nerve-stretching or ligature of the carotid. The operation of neurotomy is very simple (it can be done subcutaneously without even disfiguring the patient), but it is usually barren of results on account of the ready reunion of the divided ends of the nerves. Neurectomy is the operation generally resorted to, and portions of the nerves have been excised in every part of their course, the daring knife of the surgeon entering even as far back as the foramen rotundum at the base of the skull.

Dr. Dennis, who has lately reviewed the subject of neurotomy [1] as applied to the superior maxillary nerve, thinks that the operation offers the best chances of success when the excision is made between the spheno-palatine ganglion and the foramen rotundum. There can be no doubt, even from the merely cursory review which we have made of the surgical literature of the subject, that this operation (which is the most difficult and dangerous of all the operations for neurectomy of the trigeminus), is steadily gaining favor among practical surgeons. We must refer the reader to surgical treatises for a description of the *modus operandi* of these operations. Some of them, such as excision of a portion of the inferior dental nerve (while in the canal) are of a very simple nature, and may be performed without the possession of special surgical training.

Nerve-stretching has also been resorted to within the last few years, in cases of trigeminal neuralgia, and this method can already point to some successes. The operation has been performed upon all three branches of the nerve, and is stated to be of service even when the neuralgia has a central origin. It should be done cautiously, and the nerve not pulled upon too strongly. It is reserved for future investigations to determine the special indications for nerve-stretching and neurectomy in this form of neuralgia, and their relative merits.

Finally, we must mention the operation of ligature of the carotid for obstinate cases of this disease. This was proposed by Nussbaum, and has been performed quite frequently by this and other German surgeons, in most cases with admirable results. But the operation can only be looked upon as a last resort, and should only be performed after medicinal agents, neurectomy, and nerve-stretching have been faithfully employed.

Despite the large number of cases of trigeminal neuralgia which have been operated upon, its surgical treatment is still in a somewhat chaotic condition, and not until the surgeon enters more carefully into the minute details of the affection, will the indications for the various operations be more precisely defined.

---

[1] New York Med. Journal, 1879.

CHAPTER VII.

OCCIPITAL NEURALGIA.

Clinical History.

This term refers to an affection of either the occipitalis major[1] or minor nerves. When the auricularis magnus, subcutaneous colli, or supra-clavicular nerves are involved, the affection is known as cervico-occipital neuralgia. Occipital neuralgia is not, by any means, a very uncommon affection, while cervico-occipital neuralgia is extremely rare, and I have only seen it in combination with other varieties.

The character of the pain is entirely similar to that described in prosopalgia, but it usually presents a much less degree of severity. It shoots along the back of the head as far as the vertex, and when the occipitalis minor is involved, likewise affects the lobe of the ear. When the other above-mentioned nerves are implicated, the pain darts into the lower part of the face and chin, the front of the neck, and the upper part of the chest and shoulder on the same side. Occipital neuralgia is much more commonly bilateral than any other form of the disease. The only painful points which we have been able to detect are an occipital point, a little to the outside of the occipital protuberance, and another, at the point at which the nerve becomes superficial (between the occipital bone and the first cervical vertebra). A *point apophysaire* can be readily detected, in most cases, over the second or third cervical vertebra. During the paroxysms of pain the patients hold the head and neck as immovable as possible, because a fresh attack is very readily induced upon the slightest movement. It is important to remember, however, that the head does not assume any characteristic position in this affection. Occipital neuralgia is attended with very few complications, and the only one which I have ever noticed has been a change in the color of a lock of hair situated in the course of the nerve.

Dr. Julius Schreiber[2] reported a unique case of double occipital

---

[1] The occipitalis major nerve makes its exit from the spinal canal between the first and second cervical vertebræ, passes upward and becomes superficial at the lower posterior border of the scalp, very close to the median line. It then passes upward to supply the integument as far as the vertex. The occipitalis minor nerve is a branch of the third cervical, escapes behind the sterno-mastoid muscle, upon which it ascends to the occiput half-way between the lobe of the ear and the median line. It supplies the integument of the lateral portion of the occiput, and sometimes the median surface of the ear. The auricularis magnus is a branch of the third cervical, and supplies the integument of the mastoid process of the concha of the ear and the external auditory canal. The subcutaneous colli is also a branch of the third cervical, and supplies the integument of the upper part of the neck and under surface of the chin. The supra-clavicular nerves arise chiefly from the fourth cervical, and pass downward to supply the lower part of the neck and the upper part of the thorax.

[2] *Berl. kl.* Wschr., 1877, p. 726.

neuralgia, due to malaria, in which vaso-motor and secretory disturbances appeared in the course of the trigeminus. These complications consisted of redness of the face, injection of the conjunctiva, a profuse discharge of tears, increase in the nasal secretion, and sneezing. The patient was relieved by large doses of quinine.

Occipital neuralgia is not infrequently combined with trigeminal, sometimes one form being more severe, sometimes the other. This combination occurred in eleven cases under my observation. The pain of one variety may be so predominant that the patients fail to mention the milder pain. In a smaller number of cases, it is combined not alone with trigeminal, but also with brachial neuralgia, and in these cases we have always found marked tenderness along several of the upper cervical vertebræ.

Etiology.

In my own cases I have found no especial difference with regard to sex, the cases being distributed almost equally among both, although the females are slightly in the preponderance. Some authors have had a different experience, however, and Eulenburg states that the proportion of females to males is as five to one.

The disease chiefly affects adult life, the youngest of my patients being twenty-five years old, and the oldest seventy-six years. The majority vary from twenty-five to fifty-five years, only two of my patients being above the latter age.

The chief exciting cause appears to be exposure, and this is readily understood, as the back of the neck is peculiarly liable to these influences. The patients are also very liable to relapses from renewed exposure to such causes.

Spondylitis deformans is also a not infrequent cause of the affection. Special attention was called to this process by Dr. Julius Braun in a pamphlet which he published upon the subject several years ago.[1] It consists of a chronic inflammation of the ligaments and osseous tissues of the vertebræ (chiefly the lateral processes), attended with enlargement and deformity of the bones, and with marked tenderness of the affected tissues. Although Braun entertains exaggerated views concerning the frequency and importance of spondylitis deformans, we think it acts more frequently as a cause of occipital and brachial neuralgias than has been hitherto supposed. I can frankly state that I have probably overlooked this process in more than one instance prior to the appearance of Braun's article, and the affection is scarcely mentioned either by orthopedic surgeons or by neurologists.

In rare instances occipital neuralgia may be due to various other causes. Thus, it may arise from syphilis, malaria, caries of the first or second cervical vertebræ, tumors of the spinal cord pressing upon the nerves at their exit from the canal, direct injury from blows, or the presence of a carious tooth. Heredity exercises little influence upon its production, the only case of this nature with which I am acquainted being mentioned by Anstie; but even in this patient, brain-work and exposure are mentioned as the exciting causes.

In those cases which are combined with brachial neuralgia I have long thought that the disease was probably symptomatic of a subacute

[1] Klin. u. Anat. Beiträge z. Kentniss d. Spondylitis Deformans.

served upon the skin, such as herpes (which runs along the course of one of the superficial nerves), ordinary eczema, pemphigus, etc. Foul-looking and very obstinate ulcers may be left in the wake of these eruptions. Diminution of perspiration is the only secretory disorder of the skin which I have noticed, but Weir Mitchell has observed, in addition, excessive secretion of sweat, which was sometimes of "a disagreeable odor, like vinegar." The hair presents the same modifications which we have referred to in connection with trigeminal neuralgias, viz., atrophy, hypertrophy, change of color, and brittleness. The nails may also suffer, the rate of growth is diminished, they become more strongly curved in both directions, fissures appear in them, and they present a dirty, yellowish color.

Finally, we must refer to a peculiar affection of the joints, which may involve any of the articulations of the limb, but is usually confined to the fingers. In one case under my observation all the joints of the fingers of the left hand, together with the elbow-joint, were involved. The ends of the bones appear to be swollen, slight redness and perhaps a little swelling is sometimes observed around the joints, and they are extremely tender upon the slightest pressure in any direction. Contracture and ankylosis of the affected joints are very apt to develop under these circumstances. The following case, reported by Weir Mitchell, is a good illustration of the manner in which these changes may be combined.

Case III.—B. D. L., aged forty-three, a farmer from Maine. Enlisted, July, 1862. He was healthy to the date of his wound, received July 2, 1863, at Gettysburg. While kneeling and aiming, he was shot in the right side of the neck. He felt pain in the wound, but none down the arm. He spun around, feeling stunned, and fell on his back, not unconscious. In five minutes he arose and walked to the rear, where the wound was dressed with cold water, no splint being employed either then or later. At first all motion was lost. In an hour he could move his fingers and abduct the arm, but not flex it. He thinks sensation was perfect, except as to the ulnar distribution. Within an hour he had severe earache, and pain in the shoulder, arm, and forearm. During the second week he began to have burning pain in the hand. At this time, which probably marked the onset of neuritis, the shoulder-joint grew stiff, then the elbow, and lastly all of the fingers. This condition was excessively painful, and remained unchanged. The tremor, which is constant in the upper arm muscles, began the day of the wound, and had not ceased on his admission to our wards.

Site of wound.—On admission, October, 1863, it was noted that the ball had entered the right side of the neck, in front, three inches above the clavicle, in the outer edge of the trapezius. The missile passed downward and outward, and struck the anterior edge of the supra-spinal fossa of the scapula, five inches external to the spine of the first dorsal vertebra. Both wounds sloughed, leaving scars one and a half inches in diameter. The patient is well and florid. The shoulder is motionless from stiffness. The lower joints are alike stiff, swollen, red, and painful; the arm, semiprone and flexed, is carried across the chest, supported by the sound hand. He has slight motion throughout, but the effort causes fibrillar tremor and exquisite pain.

Sensation.—The sense of touch is everywhere good, save that there is slight numbness of the back of the hand and forearm. Some causalgia is felt in the palm, but no other pain, except on movement.

Nutrition.—The palm is thin and red and purplish, and on it the pa-

tient uses water, now and then, as a dressing; there is no atrophy; the wound is healed, but tender, as are also the upper nerve-tracks. Muscular hyperæsthesia of the deltoid and triceps is present. The nails are remarkably curved, the hair is scanty, the sweat ill-smelling and acid. The shoulder muscles alone have lost electro-muscular contractility (induction current). Under ether, the joints when moved are found to be free from well-marked organic adhesions.

Passive motion and electricity caused speedy pain in movement, and in February, 1864, he was able to move all the joints with diminished pain. The muscles were, at this time, sensitive to induced currents, and the numbness and causalgia had nearly disappeared. He was allowed a furlough, at the expiration of which he deserted.

Etiology.

Brachial neuralgia resembles sciatica with regard to etiology, in the fact that both are frequently caused by agencies which act upon the nerves after their escape from the spinal canal. Heredity plays a very unimportant part in its causation, and Anstie only mentions one case in which the patient's family presented a neuropathic history. It is so rarely due to this cause that the majority of authors do not refer to it. Salter reports several cases of this form of the disease which were due to the reflex irritation of a carious tooth, and other observers have reported similar cases. Salter believes that such patients suffer from a predisposition (either acquired or congenital) to neuralgia. Not an inconsiderable number of cases occur as complications of occipital and sometimes of trigeminal neuralgia. The brachial neuralgia then presents a lesser severity, but I have sometimes noticed that this form persists after the occipital or trigeminal affections have disappeared. Constitutional diseases, such as malaria and syphilis, are very rarely, if ever, the causes of this form of the disease. The large majority of the cases are due to local causes, which may be situated at the spinal column, in the course of the nerves, or at their peripheral distribution. The lesions of the vertebræ which may give rise to it include spondylitis deformans, caries, and carcinoma. Braun reports seven cases in which this was one of the symptoms of spondylitis deformans affecting the cervical vertebræ. This is readily determined by the local tenderness on pressure and by the presence of deformity, especially of the lateral masses. We refer to pages 144, 145 for our remarks on the diagnosis of caries and carcinoma of the vertebræ.

Brachial neuralgia may, for a considerable period, be the sole symptom of chronic cervical pachymeningitis; as a rule, the affection is then bilateral and is combined with double occipital neuralgia. It may be impossible to determine the origin of the neuralgia for a month or two, but other characteristic symptoms of pachymeningitis then make their appearance, consisting of gradually increasing motor paralysis of the arms, with contracture and progressive muscular atrophy, especially involving the hands.

As I have remarked in the chapter on the etiology of occipital neuralgia (page 133), I have long thought that this form, as well as brachial neuralgia, may be sometimes due to subacute meningitis of the cervical portion of the cord.

In the course of the nerves numerous exciting causes have been noted, such as aneurism of the subclavian artery, pressure from adjacent tumors,

wounds of various kinds (gunshot, lancet, knife), pressure of the fractured end of a bone or of superabundant callus, implication of the nerves in a retracting cicatrix, a direct blow, etc.

The exciting causes at the peripheral distribution of the nerves include the pressure of true or false neuromata, and the irritation arising from punctured wounds. Three of my cases were due respectively to the thrust of a rusty needle, a nail, and the point of a pair of scissors into the fingers. In all of these cases the neuralgia affected various nerve-branches in the forearm and shoulder.

Diagnosis and Prognosis.

Brachial neuralgia is sometimes mistaken for myalgia of the shoulders and arms, but the previous remarks which we have made on this subject will also apply here. Rheumatic inflammation of the shoulder- or elbow-joints is recognized by the existence of fever, swelling of the affected joints, local heat and tenderness, and the evident implication of the general system. There is very little danger, however, of making such mistakes unless the case is examined superficially, and too much reliance placed upon the patient's statement that he is suffering from "neuralgia," a term which, in the mouths of the laity, is expressive of very many conditions.

The main difficulty consists in a determination of the exciting cause of the disease. Whenever the affection is bilateral, and especially when it is combined with occipital neuralgia, we must pay special attention to the condition of the spinal column, and consider the possibility of the disease being secondary to spondylitis deformans, caries, or carcinoma of the vertebræ. We shall enter into this subject more in detail under the heading of intercostal neuralgia, and refer the reader to page 144 for our remarks on the subject. Chronic cervical pachymeningitis is recognized by its steady progress, the implication of the occipital nerves as well as the brachial plexuses, the continually progressing paralysis and atrophy of the muscles of the upper limbs, especially the hands, the development of contracture of the upper limbs, and the final spread of the motor and sensory disturbances to the lower limbs.

Those agencies which we have enumerated in the section on etiology as acting upon the nerves after their exit from the vertebral canal, must be determined by an accurate history of the case, and a careful examination of the arm by sight and touch. In obscure cases we must carefully examine the chest in order to determine whether the neuralgia may not be due to the pressure of a subclavian aneurism, or other intra-thoracic growth.

Treatment.

One of the essential features in the treatment of brachial neuralgia, whatever may be its origin, is the maintenance of entire rest of the parts. We not infrequently find that, after the disease has been apparently cured, a relapse readily occurs in consequence of some unusual exercise.

During a paroxysm of pain the instincts of the patient will teach him to hold the arm quiet, but he must also be enjoined to shun manual exercise for several weeks after the pain has disappeared.

Medicinal measures are of little avail in this affection. When the par-

oxysms are unendurable we must, of course, resort to the use of hypodermic injections of morphine or some of its substitutes.

Galvanism has proven by far the best remedial agent, in my hands, in the treatment of this affection. One electrode (it is immaterial which) should be applied over the lower cervical vertebræ, and the other over the course of the brachial plexus in the arm (the current should be moderately strong and not applied longer than five to ten minutes). If the neuralgia affects only the nerves in the arm, this application will be sufficient; but if the forearm is also involved, a second application should be made, one electrode being now applied to the plexus and the other to the affected nerve. The sittings may be held daily, or every other day.

Counter-irritation is usually a valuable adjuvant, especially when the disease is due to spondylitis deformans. In the latter event, the counter-irritation is best secured by the application of tincture of iodine, repeated with sufficient frequency to cause blistering; warm baths are also very serviceable in these cases. In those cases which are not due to spondylitis, we may employ emplastrum cantharidis over the vertebræ, and, in obstinate cases, over the tender spots in the course of the nerves. The actual cautery may also be resorted to, especially along the nerve-trunks. This measure sometimes produces admirable results within a very short period.

Surgical interference is frequently required in this form of neuralgia, either to relieve the nerves from the pressure of a strangulating cicatrix or of superabundant callus, to remove a neuroma, etc. I noticed as a curious fact in the three cases previously mentioned as due to the prick of a pin, blade of scissors, and nail, and in which the neuralgia appeared in almost all the nerves of the arm, that the pain was very markedly improved after several applications of the faradic current to the finger which had been the site of the injury. In some inveterate cases it will become necessary to resort to section or resection of the nerves; in such cases great caution must be exercised, and the patient very carefully examined in order to determine which nerve or nerves should be operated upon. This problem is often solved with great difficulty on account of the abundance of recurrent sensory fibres. Some successful cases of nerve-stretching for brachial neuralgia have been reported, and in these, as in all other mixed nerves, it is not at all improbable that this operation will finally supersede that of neurectomy entirely.

In the terrible form of neuralgia known as causalgia, Weir Mitchell recommends the continual application of water dressings to the affected part, and repeated blisters; hypodermic injections of morphine, preferably into the seat of pain, are indicated when the pain is intolerable.

CHAPTER IX.

INTERCOSTAL NEURALGIA.

Clinical History.

This constitutes one of the most frequent as well as the mildest forms of neuralgia. The pain is strictly confined to the course of the nerves (in the vast majority of cases to the anterior branches), and the paroxysms differ in no respect from those of other varieties of this affection. If the pain is severe the patient leans toward the affected side (usually the left), and is afraid to take a long breath. A paroxysm may be excited by coughing, sneezing, or any other sudden movement in which the thorax and abdomen take part. The pain is usually felt on the left side, from the sixth to ninth intercostal spaces. In one variety of intercostal neuralgia, which sometimes attains an intolerable intensity, the breast is the seat of pain, usually in the female, though a few cases have been reported in the male (Cooper's irritable breast). This pain is purely neuralgic in character, and does not appear to follow the definite course of any single nerve, but darts through the breast in all directions. In neuralgia of the breast, as well as in ordinary intercostal neuralgia, the integument is often extremely hyperæsthetic (in the former case over the entire breast, and in the latter along the course of the painful nerve), so that the slightest touch is unendurable; the patients frequently complain of the pain caused by the pressure of the clothes. In many cases, however, firm pressure over the painful region will produce decided relief. After a certain length of time the hyperæsthesia gives way to anæsthesia, though I have often observed the former condition even after the disease has lasted for a long period.

Puncta dolorosa are very generally observed in this disease, though it would be a mistake to believe that they constitute an absolutely essential feature. They are usually three in number, but one or even two of these may be absent. They are termed respectively the spinal point, which is situated to one side of the spinous process at the exit of the nerve from the intervertebral foramen, the axillary, situated near a line dropped from the middle of the axillary space, and the sternal point, about an inch from the sternum, or, in the nerves distributed to the abdomen, near the median line over the rectus muscle.

Herpes zoster is the only complication of intercostal neuralgia which possesses any importance. From the frequency with which herpes is referred to as a complication of neuralgia in foreign journals, it appears to be much more common in Europe than it is in our own country. According to Baerensprung it is more frequent on the right side than on the left; in very rare instances it is bilateral, and encircles the trunk like a girdle. Its appearances are similar to those observed in ophthalmic zoster, and we refer the reader to the description of the former affection on page 95.

Sometimes the eruption appears prior to the development of the neuralgia, at other times the latter ceases as soon as the herpes has developed. It is frequently a very obstinate and distressing complication, and the slightest contact with the ulcerated surface may cause excruciating agony.

Intercostal neuralgia differs somewhat from other varieties in the fact that the patients often suffer from a dull, steady pain along the nerve during the intervals between the paroxysms, and this interparoxysmal pain is often a source of considerable distress; it is sometimes so severe that the patients are compelled to restrain the movements of respiration on one side, as in cases of pleurisy with effusion.

This disease is frequently combined with trigeminal and other neuralgias, but it always follows in the wake of the latter, and does not attain an equal severity. Anstie mentions a case of death from intercostal neuralgia in a woman æt. 70 years, in whom the disease was complicated with intractable herpes ; death was directly due to exhaustion, consequent on the severe and protracted pain. This case, however, is an extremely exceptional one, and I can find no reports of similar ones.

Etiology.

The large majority of cases of intercostal neuralgia develop in women between the ages of twenty and forty years. It is, however, not infrequently met with in girls at the age of puberty, and sometimes also plays a part in the affection which we have termed the menopause neurosis (page 100).

The period in which intercostal neuralgia usually appears corresponds to the child-bearing age, and this is explained by the fact that it commonly results from excessive lactation, profuse leucorrhœa, and, as we have observed in many cases, after the patient has lost considerable blood during delivery. These patients always present evidences of anæmia, such as palpitation of the heart, shortness of breath on slight exercise, drowsiness in the day-time, and often insomnia at night, weakness, anæmic heart-murmurs, etc. We desire to call attention to the fact that this class of patients, although markedly anæmic, often present a very ruddy complexion, which is sometimes so marked that we have seen physicians make a diagnosis of congestion of the brain from the mere appearance of the face. Upon close inquiry it will be found, however, that the redness often alternates with sudden pallor, and this symptom is probably due to vaso-motor paralysis as the result of the profound anæmia.

Malaria is very rarely a cause of intercostal neuralgia, and periodicity is not well marked in this variety.

Exposure to cold, etc., also plays a very unimportant part in the etiology of the affection, and changes in temperature possess very little influence on the intensity of the pain.

The other causes of intercostal neuralgia are also infrequent, and chiefly include various affections of the vertebræ or membranes of the spinal cord. It is not uncommonly one of the first symptoms of Pott's disease, and is very generally present in the later stages when an abscess has formed which presses upon the cord or upon the roots of the nerves. It is also a frequent symptom of locomotor ataxia. But we must not confound the cincture feeling of ataxia with true neuralgia, as the former lacks the essential features of neuralgic pain. Intercostal neuralgia is

CHAPTER VIII.

BRACHIAL NEURALGIA.

Clinical History.

Although one of the more infrequent forms of neuralgia, especially in civil life, this variety often rivals trigeminal neuralgia in intensity, and is also interesting on account of the numerous and important trophic changes with which it may be complicated. The neuralgia may be strictly limited to the course of one nerve, but, in the majority of patients, several branches of the brachial plexus are involved at the same time. This is due to the fact that the nerves are situated in such close apposition in the neck and arm, and that there are frequent anastomoses between them. Henle ("Anatomie des Menschen") has furnished a plate representing the cutaneous distribution of the individual nerves of the plexus, but, for the reasons just mentioned, this is of very little service in practice.

The pains of this variety of neuralgia do not so often present the periodicity which is frequently observed in other forms of neuralgia, *i. e.*, the paroxysms appear more irregularly and, at the same time, more often than in other varieties. A striking characteristic of brachial neuralgia is the fact that the pains dart both up and down the nerves in the majority of cases, instead of merely toward the periphery, as they usually do in other forms.

Weir Mitchell,[1] to whose valuable observations we are largely indebted, has applied the term "causalgia" to one form of pain (observed in traumatic neuralgias), which he describes in the following terms: "Its favorite site is the foot or hand. The great mass of sufferers described this pain as superficial, but others said it was also in the joints, and deep in the palm. If it lasted long it was finally referred to the skin alone.

"Its intensity varies from the most trivial burning to a state of torture, which can hardly be credited, but which reacts on the whole economy, until the general health is seriously affected. The part itself is not alone subject to an intense burning sensation, but becomes exquisitely hyperæsthetic, so that a touch or a rap of the finger increases the pain. Exposure to the air is avoided by the patient with a care which seems absurd, and most of the bad cases keep the hand constantly wet, finding relief in the moisture rather than in the coolness of the application. Two of these sufferers carried a bottle of water and a sponge, and never permitted the part to become dry for a moment. As the pain increases the general sympathy becomes more marked. The temper changes and grows irritable, the face becomes anxious, and has a look of weariness and suffering. The sleep is restless, and the constitutional condition, reacting on the

[1] Injuries of Nerves, 1872.

wounded limb, exasperates the hyperæsthetic state, so that the rattling of a newspaper, a breath of air, the step of another across the ward, the vibrations caused by a military band, or the shock of the feet in walking, gives rise to increase of pain. At last the patient grows hysterical, if we may use the only term which describes the facts. He walks carefully, carries the limb with the sound hand, is tremulous, nervous, and has all kinds of expedients for lessening his pain. . . . . Motion of the part was unendurable in some of the very worst cases; but, for the most part, it did no harm, unless so excessive as to flush the injured region."

The internal cutaneous, ulnar, and radial nerves are the ones which are most frequently involved, but it is comparatively rare to find even these nerves affected to the entire exclusion of other branches. This circumstance is very readily understood, if we remember that Arloing and Tripier have shown that numerous recurrent sensory fibres, pass from the trunk of one nerve to that of another at various heights (most numerous as the nerves approach the periphery).

Brachial neuralgia is frequently associated with other varieties, especially with occipital and trigeminal neuralgias, as we have already stated in discussing these affections.

Numerous *puncta dolorosa* are mentioned by various authors, but we have found very few which are constant. A spinal point over the lower cervical vertebræ is present in almost all cases which attain any considerable degree of severity. An axillary point is also frequently observed over the course of the plexus in the axillary space. At the elbow we may find an ulnar point, between the olecranon process and the internal condyle; an external cutaneous point, above the external condyle, where the musculo-cutaneous nerve becomes superficial. The other painful points are inconstant.

The motor complications are not very important; they consist of stiffness of the limb from rigidity of the muscles, and this may even be so severe as to lead to considerable contracture. Muscular atrophy may develop, both from disuse of the limb and from an affection of the trophic fibres in the course of the nerves. Fibrillary twitchings of the muscles are sometimes observed, and even tonic and clonic convulsions of the entire limb have been noticed. These phenomena are not, however, by any means so frequent as in neuralgic affections of the lower limb, except in cases in which the disease is due to a direct wound of the nerve.

Hyperæsthesia, or more properly speaking, hyperalgesia of the skin, is a frequent complication. The integument may be exquisitely painful to the slightest touch, while tactile sensibility is unaltered or diminished. The patients also often complain of a sensation of formication and numbness.

The trophic complications are at once the most interesting and important, but they do not attain any considerable intensity unless the neuralgia is due to an injury of the nerve. The skin and its appendages are the parts most frequently involved, giving rise to what is known as glossy skin. This condition, as far as we know, is limited to the fingers and to the palm or dorsal surface of the hand. The integument of the affected portion appears to be thinner than normal, the creases are partially or entirely effaced, as if the skin were drawn tightly over the bones, and it has a peculiar shining look. Glossy skin is usually combined with hyperæsthesia of the integument, though we have not seen it attended with the burning pain which Weir Mitchell mentions (none of my cases, however, were of a severe character). Various eruptions have been ob-

served upon the skin, such as herpes (which runs along the course of one of the superficial nerves), ordinary eczema, pemphigus, etc. Foul-looking and very obstinate ulcers may be left in the wake of these eruptions. Diminution of perspiration is the only secretory disorder of the skin which I have noticed, but Weir Mitchell has observed, in addition, excessive secretion of sweat, which was sometimes of "a disagreeable odor, like vinegar." The hair presents the same modifications which we have referred to in connection with trigeminal neuralgias, viz., atrophy, hypertrophy, change of color, and brittleness. The nails may also suffer, the rate of growth is diminished, they become more strongly curved in both directions, fissures appear in them, and they present a dirty, yellowish color.

Finally, we must refer to a peculiar affection of the joints, which may involve any of the articulations of the limb, but is usually confined to the fingers. In one case under my observation all the joints of the fingers of the left hand, together with the elbow-joint, were involved. The ends of the bones appear to be swollen, slight redness and perhaps a little swelling is sometimes observed around the joints, and they are extremely tender upon the slightest pressure in any direction. Contracture and ankylosis of the affected joints are very apt to develop under these circumstances. The following case, reported by Weir Mitchell, is a good illustration of the manner in which these changes may be combined.

Case III.—B. D. L., aged forty-three, a farmer from Maine. Enlisted, July, 1862. He was healthy to the date of his wound, received July 2, 1863, at Gettysburg. While kneeling and aiming, he was shot in the right side of the neck. He felt pain in the wound, but none down the arm. He spun around, feeling stunned, and fell on his back, not unconscious. In five minutes he arose and walked to the rear, where the wound was dressed with cold water, no splint being employed either then or later. At first all motion was lost. In an hour he could move his fingers and abduct the arm, but not flex it. He thinks sensation was perfect, except as to the ulnar distribution. Within an hour he had severe earache, and pain in the shoulder, arm, and forearm. During the second week he began to have burning pain in the hand. At this time, which probably marked the onset of neuritis, the shoulder-joint grew stiff, then the elbow, and lastly all of the fingers. This condition was excessively painful, and remained unchanged. The tremor, which is constant in the upper arm muscles, began the day of the wound, and had not ceased on his admission to our wards.

Site of wound.—On admission, October, 1863, it was noted that the ball had entered the right side of the neck, in front, three inches above the clavicle, in the outer edge of the trapezius. The missile passed downward and outward, and struck the anterior edge of the supra-spinal fossa of the scapula, five inches external to the spine of the first dorsal vertebra. Both wounds sloughed, leaving scars one and a half inches in diameter. The patient is well and florid. The shoulder is motionless from stiffness. The lower joints are alike stiff, swollen, red, and painful; the arm, semiprone and flexed, is carried across the chest, supported by the sound hand. He has slight motion throughout, but the effort causes fibrillar tremor and exquisite pain.

Sensation.—The sense of touch is everywhere good, save that there is slight numbness of the back of the hand and forearm. Some causalgia is felt in the palm, but no other pain, except on movement.

Nutrition.—The palm is thin and red and purplish, and on it the pa-

tient uses water, now and then, as a dressing; there is no atrophy; the wound is healed, but tender, as are also the upper nerve-tracks. Muscular hyperæsthesia of the deltoid and triceps is present. The nails are remarkably curved, the hair is scanty, the sweat ill-smelling and acid. The shoulder muscles alone have lost electro-muscular contractility (induction current). Under ether, the joints when moved are found to be free from well-marked organic adhesions.

Passive motion and electricity caused speedy pain in movement, and in February, 1864, he was able to move all the joints with diminished pain. The muscles were, at this time, sensitive to induced currents, and the numbness and causalgia had nearly disappeared. He was allowed a furlough, at the expiration of which he deserted.

Etiology.

Brachial neuralgia resembles sciatica with regard to etiology, in the fact that both are frequently caused by agencies which act upon the nerves after their escape from the spinal canal. Heredity plays a very unimportant part in its causation, and Anstie only mentions one case in which the patient's family presented a neuropathic history. It is so rarely due to this cause that the majority of authors do not refer to it. Salter reports several cases of this form of the disease which were due to the reflex irritation of a carious tooth, and other observers have reported similar cases. Salter believes that such patients suffer from a predisposition (either acquired or congenital) to neuralgia. Not an inconsiderable number of cases occur as complications of occipital and sometimes of trigeminal neuralgia. The brachial neuralgia then presents a lesser severity, but I have sometimes noticed that this form persists after the occipital or trigeminal affections have disappeared. Constitutional diseases, such as malaria and syphilis, are very rarely, if ever, the causes of this form of the disease. The large majority of the cases are due to local causes, which may be situated at the spinal column, in the course of the nerves, or at their peripheral distribution. The lesions of the vertebræ which may give rise to it include spondylitis deformans, caries, and carcinoma. Braun reports seven cases in which this was one of the symptoms of spondylitis deformans affecting the cervical vertebræ. This is readily determined by the local tenderness on pressure and by the presence of deformity, especially of the lateral masses. We refer to pages 144, 145 for our remarks on the diagnosis of caries and carcinoma of the vertebræ.

Brachial neuralgia may, for a considerable period, be the sole symptom of chronic cervical pachymeningitis; as a rule, the affection is then bilateral and is combined with double occipital neuralgia. It may be impossible to determine the origin of the neuralgia for a month or two, but other characteristic symptoms of pachymeningitis then make their appearance, consisting of gradually increasing motor paralysis of the arms, with contracture and progressive muscular atrophy, especially involving the hands.

As I have remarked in the chapter on the etiology of occipital neuralgia (page 133), I have long thought that this form, as well as brachial neuralgia, may be sometimes due to subacute meningitis of the cervical portion of the cord.

In the course of the nerves numerous exciting causes have been noted, such as aneurism of the subclavian artery, pressure from adjacent tumors,

and at the inner side of the knee-joint. In rare instances herpes zoster occurs in crural neuralgia. In severe cases the limb is held motionless as in sciatica, to prevent an increase in the severity of the painful paroxysms. It is unnecessary to give the symptoms of each form in detail; they merely differ with regard to the distribution of the pain.

A good example of this variety of neuralgia, and which at the same time illustrates the frequent dependence of the disease upon other affections, is shown in the following case, which I saw in consultation with Dr. John Munn, of this city, to whose kindness I am indebted for the following notes:

CASE V.—S. L., æt. 23 years; a kept mistress; no neuropathic tendency in the family. The patient took cold during her first menstruation (at the age of fourteen) and this was immediately followed by severe pain in the distribution of the middle and external cutaneous nerves of the right lower limb. The pain was sharp and excruciating, much worse at night, and lasted for three months. After this, "catching cold" would cause the pain to reappear; she was never worse during menstruation. In 1877, her physician thought she had some uterine trouble, and sent her to Hot Springs, where she had a very severe attack of neuralgia. In 1878 she was treated by Drs. Briddon and Seguin during a very bad attack lasting nearly three months, but with very little benefit from the treatment (morphine and galvanism). The patient came under Dr. Munn's care on April 1, 1879. Upon examination, signs of endocervicitis were found present, and the uterus was slightly retroverted and a little lower down in the pelvis than normal. There was some tenderness on pressure to the right of the uterus anteriorly. Applications were made to the os uteri for a month, with marked improvement in the condition of the organ; the neuralgic symptoms improved at the same time, although they were not treated directly.

June 1st.—The pain again returned in a very severe form, after indulgence in sexual intercourse, which had been interdicted. I saw the patient in consultation on June 15th; the pain was of an excruciating character, and confined to the anterior and lateral aspects of the right thigh, extending nearly to the knee; it was of a shooting character, but unattended with hyperæsthesia or trophic disturbances; no painful points along the spine. Cervical catarrh again present, and tenderness felt upon pressing deeply into the abdomen on the right side over the brim of the pelvis about midway between the symphysis and sacrum. Pressure at this point sometimes gave rise to shooting pains down the thigh.

I advised the continuance of the local treatment of the uterus, together with the internal administration of Fowler's solution and aconitia, gr. $\frac{1}{100}$ night and morning, in increasing doses until the physiological effects are produced.

July 1st.—The pain has ceased and her condition is excellent; treatment with arsenic continued. The patient then went to Saratoga for the summer, and remained well, with the exception of a few occasional twinges, until October 1st. The pain now returned in a very violent form, and persisted with a few intermissions until November 11th. She was then seen by Dr. Thomas in consultation, who found slight retroversion of the uterus. Dr. Thomas believed that the neuralgia was due to some obscure neurosis, and that the uterine disorder was only a partial cause; he advised replacement, but expected no good from it.

Toward the end of November I again saw the patient in consultation.

The pain was now of a frightful character, and could only be controlled by immense doses of morphine, as much as 100 minims of Magendie's solution having been administered on one occasion within three hours. The patient was in an extremely hysterical condition, which was partly due to continual worry from the belief that she was about to be discarded by her lover.

A large number of remedies had been resorted to in succession (bromide of potassium, atropine, aconitia, strychnine, blisters, etc.) in heroic doses, but not the slightest benefit was obtained. A tolerable amount of relief was only afforded by hypodermics of morphine. On December 9th the patient started for Baltimore, the pain having subsided somewhat during the last week; this is probably due to the fact that the cause of her mental worry has been removed. Since that time the patient has felt perfectly well.

This case is interestisg from several points of view. In my opinion the chief cause of the protracted character of the disease was the hysterical condition, induced by leading a life of luxury, with no object in view save pleasure. I am also convinced that the uterine disorder was a potent etiological factor; it is quite probable that the patient suffered from mild pelvic peritonitis during her first menstrual epoch, and that some slight inflammatory products remained in the pelvis and acted partly as the exciting cause of the neuralgia.

Etiology.

This form of neuralgia is so infrequent, that very little is definitely known with regard to its etiology. It may be due to carcinoma, spondylitis deformans, or caries of the lumbar vertebræ, or to growths of various kinds in the pelvis. In one of my patients crural neuralgia followed a forceps delivery. In a considerable proportion of the cases it appears to be of a reflex nature, due, as Mauriac has shown, to orchitis or epididymitis; it may also be connected with uterine disease, as in the case reported above. I have also seen a few examples of ilio-inguinal neuralgia in male patients who were victims of "nervous debility" and the general lowering of morale which occurs when the individual is suffering from seminal emissions and its accompanying train of symptoms. This is not infrequently combined with neuralgia of the testis, an extremely distressing malady. Neuralgia of the obturator nerve has been observed in obturator hernia, and the occurrence of the former should always lead to a careful examination with regard to the presence of the latter. Perhaps the majority of cases of lumbar neuralgia are found to develop during severe paroxysms of sciatica, and in this event the middle and internal cutaneous nerves (branches of the crural) are usually the ones involved. In some instances, indeed, the lumbar neuralgia which has begun under such circumstances acquires an independent existence, and becomes even more formidable than the primary sciatica. Finally, a small number of cases have been reported, in which the disease was apparently due to exposure or to overexertion of some of the lumbar muscles.

Diagnosis.

Lumbar neuralgia must be differentiated from myalgia of the lumbar muscles or lumbago; the character of the pain, its distribution, the inability to move the affected muscles without producing severe suffer-

ing, and the consequent fixed position of the trunk, and the absence of puncta dolorosa, are sufficient to exclude neuralgia. In rare cases, however, lumbago is combined with lumbar neuralgia, as in a patient recently under my observation, in whom lumbago of long standing became complicated with neuralgia of the ilio-inguinal nerve. Myalgia of the anterior thigh muscles may also be mistaken for crural neuralgia, but attention to the symptoms mentioned above will prevent error.

The latter form may also be mistaken for morbus coxæ or neuralgia of the hip-joint (Brodie's joint), but we shall enter into this subject at some length under the head of sciatica (page 158), which is also liable to be confounded with these two affections.

One of the main indications in making the diagnosis is to determine the nature of the primary lesion. In all cases in which a definite cause is unknown, the lumbar spine and pelvis should be subjected to careful physical exploration. The etiological factors vary but little from those which we have described with reference to neuralgias of the upper limb (with the exception of its occurrence in uterine affections), so that we may refer to the remarks on diagnosis under that head. Before affirming the existence of any connection between a uterine affection and some form of lumbar neuralgia, we should satisfy ourselves that the latter improves, *pari passu*, with the former, and has followed it in point of time. We must not forget, also, that both affections may be the expression of depressed vitality of the general system.

Treatment.

Fortunately this disease does not often assume extreme severity. The indications for rational treatment can very rarely be met. In cases of spondylitis or caries of the spine, and in uterine disease, the appropriate treatment for these affections may prove useful, but in the majority of cases we must rely chiefly on symptomatic measures.

Counter-irritation, in the form of fly-blisters applied over the painful spots at the exit of the affected nerve from the lumbar spine, is of decided advantage. It should be applied more vigorously than in intercostal neuralgia, as the nerves are farther removed from the integument. The actual cautery may also be used, preferably along the course of the cutaneous distribution of the nerve. The chief reliance must be placed upon the use of morphine, when the pain is intolerable, and upon the steady application of the continuous current. In employing the latter one electrode is placed at the exit of the nerves from the lumbar spine, the other upon some portion of their external course. The current should possess considerable intensity, as the nerves are situated deeply, and are therefore not readily affected by electricity. I should also mention that in the case of lumbago and ilio-inguinal neuralgia to which I have referred, and which had caused constant and severe suffering for a year, relief was very quickly obtained by the use of the faradic current, applied over the painful parts. Internal remedies have not appeared to be of much service. In my own hands the greatest amount of relief has been obtained from the use of strychnia, beginning in doses of gr. $\frac{1}{4}\frac{1}{8}$, t.i.d., and gradually increasing until physiological effects are obtained. Sometimes, as occurred in the case reported on page 148, abundant opportunity will be afforded us to experiment in succession with a considerable proportion of *the neurotics* contained in the materia medica.

CHAPTER XI.

SCIATICA.

Clinical History.

This form of neuralgia ranks next to that of the trigeminus in importance and interest, on account of its severity and frequency. The disease usually begins with prodromata, consisting of a feeling of heaviness in the limb, numbness and tingling, or a sensation of coldness. Exceptionally a paroxysm begins suddenly, and in one instance I saw a patient (who had never suffered from any form of neuralgia previously), in whom one of the most violent paroxysms which I have ever seen, developed immediately after rising from a kneeling posture. The pain is of a markedly lancinating, darting character, and may involve the entire course of the nerve; it usually darts toward the periphery, but sometimes shoots up and down the nerve with intolerable violence. It is sometimes so intense as to cause even the bravest patient to cry out in agony and to roll on the floor in despair. In one of my cases the pain was so severe, notwithstanding the hypodermic injection of a very large dose of morphine, that I could only restrain the patient by main force from committing suicide. But these cases are very rare, nor do we find, save in exceptional instances, that the hysterical, broken-down condition of mind is produced which is met with in bad cases of trigeminal neuralgia. In the beginning of the affection the pain is very commonly limited to the upper part of the course of the nerve, and after a while it spreads to the lower branches. But the reverse also holds good, and individual branches (perhaps such a small twig as the plantar nerve) may be alone affected throughout the entire course of the disease. If the patient is intelligent and a good observer, he will be able to trace the course of the nerve with his finger, and this is sometimes done as accurately as by a well-informed anatomist.

Puncta dolorosa are observed in nearly all cases. The most usual sites in the order of frequency are: a *gluteal* point, beneath the gluteal fold, half-way between the trochanter major and the tuber ischii; a point at the emergence of the nerve from the sciatic foramen; a point at the posterior superior spinous process of the ilium; one or two *popliteal* points, at either side of the popliteal space, immediately within the hamstrings; a *fibular* point, behind the head of the fibula; two *malleolar* points, at the posterior part of each malleolus. The *point apophysaire* is not by any means so common as the puncta dolorosa, and its distribution varies somewhat, tenderness being sometimes appreciable over one or more of the sacral vertebræ, and at times over the first and second lumbar vertebræ. We not infrequently find, especially in those cases in which there is considerable dull pain in the intervals of the paroxysms, that the entire length of the nerve is sensitive to pressure.

The pain is usually confined to one nerve, but in some cases both are involved, even when the disease is not connected with any affection of the spinal cord. But we should always be careful to exclude diseases of the spinal cord or its membranes whenever we are brought in contact with a case of bilateral sciatica.

Patients suffering from severe forms of this disease are generally confined to their back. The slightest movement of the limb, the act of defecation, coughing, sneezing, sitting down, and sometimes the mere contact of the bedclothes with the affected leg will prove sufficient to produce a paroxysm of pain. In severe cases, the pain is often irradiated to the distribution of other nerves, usually the crural, though even more distant nerves, such as the trigeminus, may be thus affected. The irradiated pain, however, never attains the severity of the primary affection.

When the pain is severe, disturbances of sensation are usually well marked. In the beginning of the attack we generally meet with hyperæsthesia of the skin, which may be either localized in small spots or diffused over the entire distribution of the nerve. In some cases the hyperæsthesia is so well marked that the slightest contact with the skin is agonizing to the patient, so that he cannot even bear the weight of the bedclothes. After the disease has lasted for a long time anæsthesia develops and is sometimes very pronounced. Very frequently, also, we may notice spots of hyperæsthesia and of anæsthesia on the limb at the same time.

Sciatica presents important and interesting motor complications, which are usually manifested only in cases that attain considerable severity. Fibrillary twitchings are not uncommonly observed, especially in the calf muscles and those of the back of the thigh. At times these muscular twitchings become more widespread, and the whole limb is thrown into clonic convulsions; these are only observed at the height of the paroxysm, and soon subside. In one extremely severe case the whole body was thrown into violent convulsions (not preceded by tonic spasm and unattended with loss of consciousness), but, as the patient had been drinking whiskey very freely (this appeared to be the exciting cause of the sciatica), I am unable to state positively whether the convulsions were the result of the neuralgic affection or not.

Contracture sometimes occurs at the knee-joint. We do not now refer to the voluntary stiffness of the limb assumed by the patient in order to prevent pain, but to a rigidity of the hamstring muscles, which cannot be overcome by any reasonable exercise of muscular power, either on the part of the patient or the physician. The knee may be bent at quite a sharp angle, and the contracture may persist for several weeks or even longer; it does not disappear during sleep.

The gait in severe cases is peculiar. The patient in walking keeps the limb slightly flexed at the knee, and walks on the toes of the affected foot. The gait is stiff and awkward, and the patient favors the sound limb. The entire muscular tissues of the limb sometimes atrophy. The amount of wasting is usually slight, and is due to the comparative disuse of the limb occasioned by the severe pain. But, in exceptional cases, the muscular atrophy is excessive, and cannot be accounted for in this manner. We must therefore fall back upon the supposition that it is a trophic change, due to the implication of the trophic fibres contained in the nerve-trunk by the morbid process which has given rise to the neuralgia.

Hypertrophy of the muscles of the thigh and calf has also been observed in one case.

The following history, the notes of which were obtained through the kindness of my house physician, Dr. Wyman, exemplifies most of the motor disorders observed in severe forms of sciatica.

CASE VI.—Wm. Schmidt, æt. 30 years; single; admitted to Randall's Island Hospital, September 25, 1879; family history good, never had rheumatism, denies venereal. The patient always enjoyed good health prior to this disease. In November, 1877, he was shipwrecked, and compelled to remain in the water for twenty-four hours. About six weeks after this exposure he began to have pain in the left gluteal region, which extended down the back of the thigh, following the course of the sciatic nerve. The pain was increased very much during bad weather, so that he often had to take to his bed. Not obtaining relief, he was admitted to the Homœopathic Hospital, Ward's Island, and after remaining there three months unimproved, the surgeon cut down upon the sciatic nerve and stretched it. After this operation the patient says his pains were very much increased, and he was never free from them except while under the influence of an opiate. He was discharged unimproved at the end of six months.

March 29, 1879, he was admitted to Charity Hospital, Jersey City, where the wound made by the operation for nerve-stretching healed, but the sciatica did not improve. He was treated with the actual cautery along the course of the nerve; the patient remained there two months, and was then admitted to Randall's Island Hospital.

On admission his general condition was poor; he complained of intense paroxysms of shooting pain (which forced him to whimper) along the entire course of the left sciatic nerve, and which were greatly heightened by the slightest movement of the limb. The pain was so severe that he was compelled to keep to bed constantly. There were frequent fibrillary twitchings in the muscles of the back of the thigh, and marked atrophy of the muscles of the affected limb (unfortunately no record was kept of the difference in the measurements of the two limbs, but I have a distinct recollection that the affected thigh was at least one and a half to two inches less in circumference than the healthy one).

Aconitia, gr. $\frac{1}{100}$, t.i.d., was ordered, to be gradually increased until the physiological effects were obtained. This treatment was continued until October 26th, and produced slight improvement.

October 27th.—Ordered strychniæ sulph., gr. $\frac{1}{32}$, t.i.d., to be increased one dose daily until the full effects of the drug were obtained.

November 5th.—Since the administration of the strychnia, his general condition has improved very much; he has a fine appetite, and has gained in flesh. He is often comparatively free from pain, and rests well at night. At varying intervals, which have been longer in duration since the use of strychnia was begun, severe paroxysms were felt. At such times the hypodermic administration of morphia has been resorted to with good effect. He is at present taking gr. $\frac{1}{4}$ of strychnia daily; this treatment was continued until November 29th, with several intermissions of a few days each, whenever the physiological effects of the drug were too pronounced. The duration of the intervals between the paroxysms was considerably increased, but the pain felt during the latter was still extremely severe.

December 2d.—Ordered atropiæ sulph., gr. $\frac{1}{100}$, t.i.d., to be increased one dose daily.

December 6th.—The patient received six doses of atropia (gr. $\frac{1}{\text{[illegible]}}$)

yesterday; this morning he has dryness of the throat, and disturbance of vision; pupils dilated. He has not had a paroxysm of severe pain since the administration of the drug was commenced. The paroxysms then appeared again, and the atropia was continued until January 10th, with several intermissions of a few days each. The patient now feels much better, except when he moves the affected limb. For the past few weeks the limb has been contractured at the knee-joint; the contracture could not be overcome by the patient or by myself, on account of the severe pain to which such attempts gave rise. To relieve this symptom, hot sitz-baths were ordered.

January 6th.—The patient experiences a good deal of relief from the baths. The hamstring muscles, which were formerly contracted, are now becoming relaxed, so that the leg can be extended without producing pain.

January 20th.—The patient is improving rapidly; he is up and about the ward, but has to walk with crutches.

January 28th.—Crutches taken away; patient walks with a stick, and has slight pains in locomotion.

February 2d.—Patient walks now without difficulty; the pains have entirely disappeared. The muscles of the affected limb have regained the greater part of their natural strength (no measurements were taken). The patient's general condition is excellent. Discharged cured, and has since gone to work.

The other complications of sciatica are infrequent and unimportant. In some cases the limb is hot and perspiring, in others the skin is dry and brittle; the integument may be of a uniform red color or mottled in appearance; sometimes it is paler and cooler than the other limb. Glycosuria constitutes the most interesting of the vaso-motor complications of sciatica, although very few observations have been made on this subject.

Schiff had shown that section of the sciatic or other large nerve-trunks in certain of the lower animals was capable of producing mellituria, and this physiologist, as well as others who have corroborated his experiments, have regarded the symptom as due to reflex paralysis of the vaso-motor nerves supplying the liver. Braun [1] found several cases of sciatica in which sugar was demonstrable in the urine, and the correctness of his observation has been since verified by Rosenbach. Though the histories of these cases are unsatisfactory, it appears that they never lead to true diabetes, but only to a temporary mellituria. The presence of sugar in the urine in such cases has been explained in the same manner as Schiff's cases of experimental diabetes, but this view is purely hypothetical, and further investigation may cause a change of opinion in this respect. The subject is very interesting, and one which is worthy of thorough and continued research.

Etiology.

The etiology of sciatica is well defined by Eulenburg when he calls it "the type of peripheral, accidental neuralgias."

Anstie observed four cases in which heredity played an important part, but this is probably an exceptional experience. Many authors scarcely mention heredity as one of the etiological factors in this affec-

[1] Systematiches Lehrbuch der Balneotherapie, 1868.

tion, and all are agreed that it has very little influence in this direction.

The disease is very infrequent in childhood, and Soltmann,[1] the latest writer on the subject, has not met with a single example at this period of life. The majority of the cases occur between the ages of twenty to fifty years, but the affection develops not infrequently long after the latter age, three of my cases beginning at the ages of sixty-eight, seventy, and seventy-four years respectively.

According to the united testimony of the majority of authors, the male sex is much more subject to sciatica than the female, and in my own cases the proportion has been as one to two and a half. Arnoldi, who has furnished the largest statistics on the subject, finds that females were affected almost as frequently as males (172 males, 166 females), but this statement is entirely opposed to the common experience of physicians.

The majority of the causes which give rise to sciatica are those which act upon the nerve after its exit from the spinal canal. The lesion may, however, be situated in the bones of the spinal column or within the canal. This category includes spondylitis deformans, cancer of the vertebræ, or the presence of gummata which have grown from the dura mater or from the vertebræ themselves. These various lesions act by simply producing pressure upon the roots of the nerve, and the neuralgia caused thereby is of a peculiarly lancinating character. In very rare instances false and even true neuromata have been found within the spinal canal and growing upon the cauda equina. It may also be produced by numerous processes situated within the pelvic cavity, both in the male and female. Any affection which interferes with the return of venous blood from the pelvis will predispose to its development. The sciatic nerve is surrounded by a large number of veins, the greater part of which constitute the hemorrhoidal plexus. These veins are large, wide-meshed, and possess no valves, so that an obstacle to the flow of blood in them will very readily give rise to a varicose condition, and consequently to pressure upon the nerve. Such an obstacle may be due to cirrhosis of the liver, or to any other disease of that organ or of adjacent parts which will interfere with the portal circulation. Within the pelvis itself this condition may be due to pregnancy, to the use of forceps during delivery, to the growth of intra-pelvic tumors, to exudations into the broad ligaments or into Douglas' cul-de-sac, or to an accumulation of hardened fæces in the rectum. It has been doubted by some writers whether the latter cause is ever capable of producing pressure upon the sciatic nerve, but a case which was recently under my observation is, to my mind, conclusive in this regard. The patient in question had had no evacuation for twelve days, and complained of formication and anæsthesia, which was confined to the exact distribution of the left sciatic nerve. The exhibition of a purge, which produced a free passage, caused these symptoms to disappear entirely. It is difficult to determine, however, whether the causes we have just mentioned act by pressing directly upon the nerve or by producing dilatation of the hemorrhoidal plexus and secondary pressure in this manner.

In the course of the nerve outside of the pelvic cavity, there are also numerous lesions capable of producing sciatica. This category includes injuries to the nerve, such as those produced by gunshot wounds (which are very rarely met with except in military surgery), wounds made by the

[1] Gerhardt's Handbuch der Kinderkrankh.

lancet in venesection (not very infrequent formerly, but never observed at present), blows from blunt instruments, falls on the buttocks, popliteal aneurism, neuromata, syphilitic and other tumors of the nerve itself or of surrounding tissues. Those cases which are attributed to severe muscular strain should be included under the head of injury to the nerve. Some writers are skeptical with regard to the efficacy of this cause, but one of the most severe cases of sciatica which has come under my observation occurred in a robust farm-hand, immediately after lifting a heavy beam. At the time of the accident he "felt something crack" in the lower part of the back, and immediately began to suffer from intense pains along the sciatic nerve which resisted all treatment for upward of a year.

Cotugno had called attention to the fact that sciatica prevails endemically in the neighborhood of Naples, and that this circumstance is explained by atmospheric influences. There is no doubt that the affection is more apt to develop in moist, windy weather, and the severity of an attack always increases under such conditions. Exposure to atmospheric changes also explains the comparative frequency of sciatica in those whose occupation requires them to be exposed to all kinds of weather. I have noticed this especially in coachmen, but in this class two causes may cooperate in the production of the disease, viz., the exposure to changes of weather, and the constant sitting position which the patients are compelled to assume, and in which the sciatic nerve may be pressed upon by the projecting seat.

Sciatica is not often due to constitutional causes, and differs in this respect from other forms of neuralgia. As a rule the patients are not anæmic at the beginning of the disease, although this condition is readily produced after the sciatica has lasted for some time, on account of the depressing influence of the pain, the lack of exercise, etc. But we find that sciatica not infrequently develops in old age, after degeneration of the vessels has begun, although this is not of such frequent occurrence, by any means, as in the case of trigeminal neuralgia. In such an event the disease may continue uninterruptedly until death.

Malaria is not so often a cause of sciatica as it is of other neuralgias, and is not even mentioned by many neurologists as an etiological factor. According to Schramm, malarial sciatica usually affects the entire trunk of the nerve (usually the right), and in rare cases is bilateral. In one case under my observation the pain took the place of the chill, and was confined to the plantar branch of one nerve. It generally assumes the quotidian, but sometimes the tertian type. We must remember, however, that malarial neuralgia may sometimes be continuous, and on the other hand, that the pain may assume a pure intermittent type, although the neuralgia is due to some other cause, or may even be symptomatic of an organic lesion of the nerve. We should, therefore, not make a diagnosis of malarial sciatica unless other evidences of malarial infection are present, or unless quinine exercises a specific influence upon its course.

Syphilis is also an infrequent cause of this disease, though it would appear from recent investigations that it is produced more often in this manner than was formerly believed. Fournier called attention to the fact that sciatica may be caused by the mere presence of the syphilitic virus in the blood during the early secondary stage. Its development in this stage must, however, be very rare, since it is hardly ever observed even by specialists in venereal diseases. Dr. R. W. Taylor, of this city, *who has written* an interesting article on syphilitic sciatica in the *New*

York Medical Journal, March, 1880, has only met with two cases in the secondary stage, and Dr. Keyes informs me that he has not observed a single example. In the large majority of cases, however, it is due to syphilitic changes in the nerve itself, or to irritation or compression of the nerve by gummy growths in the adjacent tissues.

Considerable doubt has been cast upon the reported cases of alternation of acute articular rheumatism and sciatica. No instances have come under my own notice, and the cases reported in the journals are described so unsatisfactorily that it is difficult to arrive at a definite conclusion in the matter. There can be no doubt, however, that a causal connection between these two affections does exist in rare instances.

Fournier has reported seven cases of sciatica occurring during the course of gonorrhœal rheumatism (which is not a true rheumatism), and others have been since observed. The pain in this variety usually disappears with the rheumatic affection.

Sciatica has also been attributed to gout, but it is doubtful whether this does not act merely as a predisposing cause on account of the abdominal plethora which is so common in the latter affection.

I also wish to call attention to the relation of delirium tremens to sciatica. Three of the severest cases of sciatica which have come under my observation occurred during the first period of a mild attack of delirium tremens, in patients who had never been previously subject to neuralgia. In all three patients the disease was of very short duration (in one it only lasted twenty-four hours), but during this time their tortures were terrible. Relief could only be obtained by the hypodermic injection of large doses of morphine.

Reflex irritation possesses very little efficacy in the production of sciatica. It sometimes develops in the course of other neuralgias, such as trigeminal or brachial, but it is difficult to determine whether this is the result of a reflex irradiation of the pain, or the expression of a general cause. In the larger number of cases the source of reflex irritation appears to reside in the genital organs. Mauriac has shown that it may occur during the course of gonorrhœal epididymitis. It has also been observed as the result of stricture of the urethra or of a stone in the bladder. It may be due to a uterine disorder which has not produced any pressure upon the nerve. A few apparently authentic cases have been reported in which sciatica was caused by the presence of worms in the intestines.

Diagnosis and Prognosis.

Sciatica is perhaps more frequently mistaken for myalgia of the muscles of the loins and thigh than for any other form of disease. But the latter affection usually involves not alone the muscles of the posterior, but also of the anterior part of the thigh; in other words, the pain is not strictly limited to the distribution of the sciatic nerve. *Puncta dolorosa*, which frequently form such a prominent part in the history of sciatica, are never present in myalgia, and the pain and tenderness are diffused in the latter affection, while in sciatica, as Valleix expresses it, the patient marks out the course of the pain with the tip of the finger. The pain of myalgia is always absent when the limb is kept perfectly quiet, while that of sciatica, although aggravated by movement, also develops when the leg is motionless. We have previously referred, in the general chapter on diag-

nosis, to the characteristics of myalgic pain, and these alone will usually be sufficient to enable us to make a differential diagnosis.

Locomotor ataxia is often distinguished with difficulty from sciatica. Cases have been reported in which ataxia was diagnosed, although no other symptom beyond that of the ataxic pains had been observed for a period of even twenty years, and we can therefore readily conceive that a differential diagnosis is often very difficult and sometimes even impossible. The pains of ataxia are invariably bilateral, and always of the most marked lancinating character. They are not confined to one part of the course of the nerve, but affect indifferently sometimes one, sometimes the other portion, and are frequently situated deeper than ordinary sciatica, involving not only the muscles, but apparently also the bones, and even the various joints. They are often accompanied by a sensation of pressure (cincture feeling) around the ankles, calves, thighs, or lower part of the chest or abdomen. The patient may experience difficulty in voiding the urine, or suffers from incontinence. Another symptom of considerable value (though it is very frequently absent) is irregularity of the pupils. The most important diagnostic sign of ataxia is the absence of the patellar tendon reflex, which is almost invariable (I found it present, however, in an undoubted case of ataxia in the paralytic stage). The patellar reflex is obtained by directing the patient to cross one knee over the other, and allow the pendent leg to hang loosely. If the ligamentum patellæ, which is thus placed upon the stretch, be then tapped smartly with the finger or with a pleximeter, a sudden forward movement of the entire leg will be produced in healthy individuals.[1] In ataxia this phenomenon becomes lost, even before any ataxic movements are visible in the gait.

Sciatica is sometimes, though rarely, mistaken for hip-joint disease and *vice versa*, but this error can only be made during the first stage of morbus coxæ. The latter disease is accompanied by drooping and partial effacement of the gluteal fold on the affected side, the limb appears to be somewhat shortened, and tenderness is manifested when the joint surfaces are brought in contact with one another. There is interference with the perfect mobility of the limb at the hip-joint, and this important point is determined by placing the patient on a horizontal even surface, the healthy limb being also on the level surface, and the diseased limb remaining flexed; it will then be found that the pelvis is at right angles to the horizontal limb, and that the lumbar spine is in contact with the surface upon which the patient rests. As soon, however, as an attempt is made to carry the flexed limb into the same position as the other, the pelvis will be found to tilt upward, carrying the lumbar spine along with it, so that the hand can be introduced underneath the vertebræ. These symptoms are always present in hip-joint disease, and a careful examination will, therefore, enable us to make a differential diagnosis.

Hysterical joints (Brodie's joint) are sometimes differentiated with difficulty from sciatica when the hip is involved. This affection is characterized by extreme tenderness of the joint to slight pressure, while firm compression is often very well borne. Painful points can be usually detected around the joint. Contracture of the hip (which disappears during chloroform narcosis) is a common feature, and may lead to a suspicion of inflammation of the joint. One of the most important differential diagnostic points is the fact that hysterical joints, unlike sciatica,

[1] I have recently noted its absence in two of my students, who were perfectly healthy.

almost invariably appear in young females in whom other well-marked symptoms of hysteria are also present.

An important feature in the diagnosis of sciatica is the localization of the primary lesion. It is impossible for us to enter into this subject *in extenso*, and we can only refer to our remarks on the etiology of the disease. But we desire especially to impress the necessity of a careful exploration of the pelvis (by palpation, vaginal and rectal exploration), whenever we have to deal with an obstinate case which resists ordinary methods of treatment. Entirely unlooked for and very valuable data will sometimes be revealed from a compliance with this rule. In every case of double sciatica we should not alone make a pelvic exploration, but also carefully examine the condition of the vertebral column.

Treatment.

The exciting cause of sciatica can sometimes be removed by a resort to surgical measures, such as the extraction of foreign bodies, the extrication of the nerve from constricting cicatrices, or from exuberant callus formed after fracture of the pelvis or long bones of the limb, ligature for aneurism of the popliteal artery, etc. When the affection is due to the pressure of intra-pelvic exudations, etc., great relief is sometimes afforded by the successive application of fly-blisters to the iliac fossa in combination with palliative doses of morphine and careful attention to the regulation of the bowels. In a large number of cases we are powerless to remove the exciting cause, even though it be well known, and we are then compelled to rely exclusively on the use of palliative measures.

In ordinary cases of sciatica it is well to begin treatment with the administration of a saline cathartic, even when the patient states that the bowels are regular. We often find that the quantity of fæces voided is insufficient, although the patient goes to stool every day, and an aloes pill, taken at bedtime, is very serviceable under such circumstances. The diet should be carefully regulated, and fatty or very feculent substances allowed only in moderation. The patient should be examined with regard to the presence of hemorrhoids, and appropriate treatment adopted. These measures are especially indicated in cases in which the patient has recovered from one attack and is in danger of a relapse.

When the pain of a paroxysm is unendurable the hypodermic administration of morphine is indicated in doses adapted to each individual case. Many authors recommend that the injections be made over the track of the nerve, or even into its tissue; but apart from the fact that an injection into the nerve itself is an operation which can only be performed with great difficulty with an ordinary hypodermic needle, I have been unable to observe any special beneficial effects from local injections.

Dr. Comegys [1] has obtained benefit from hypodermic injections of ether during the attack, and regards this as a curative measure.

The curative treatment of sciatica consists in the use of counter-irritants, the administration of certain nervines, the application of galvanism, and surgical interference.

Fly-blisters constitute the most serviceable form of counter-irritation. They are best applied over the painful spots, beginning above and passing to the one below when the sore caused by the blister previously applied

[1] Cincin. Lancet and Clinic, 1879, ii. 10.

PERIPHERAL PARALYSIS.

CHAPTER I.

CLINICAL HISTORY.

PARALYSIS is a loss of muscular power due to an interference with the transmission of nerve force from its site of development in the central nervous system to the termination of the nerve-fibres in the muscles. The term paresis simply implies a partial paralysis.

Peripheral paralysis is that variety in which the interference with the transmission of nerve force is due to some lesion which is operative between the exit of the nerves from the nerve-centres, and their termination in the muscular fibres. The affection may develop slowly or suddenly, according to the nature of the underlying cause. Thus, a sabre-cut which entirely divides the filaments of a motor nerve must produce immediate paralysis in its distribution. The gradual compression of a nerve, however, by a slowly developing neoplasm in its neighborhood will produce paralysis very slowly, and the gradually developing loss of power may be preceded by various symptoms of irritation on the part of the nerve, as we shall show hereafter.

All parts of the muscular system may be subjected to paralysis; changes in the appearance of these parts are due to the fact that the antagonist muscles have no opposing force to overcome, and therefore exercise traction upon the paralyzed ones. Thus, in paralysis of the seventh nerve, the face is drawn to the sound side because the healthy muscles no longer meet with any opposition to their tonic contraction.

The distribution of peripheral paralysis is entirely different, in the vast majority of cases, from that due to diseases of the brain or spinal cord. In the former the paralysis is situated, as the very term implies, in the distribution of individual nerves or of a number of nerves. Thus, only a single muscle may be paralyzed, as for instance in a lesion of the posterior thoracic nerve which supplies the serratus magnus muscle. When the paralysis is due to some affection of the brain or spinal cord, however, the muscles affected are those which belong together functionally, and, as a rule, a limb is paralyzed as a whole. Thus, in cerebral affections, the paralysis usually assumes the hemiplegic, and in disorders of the spinal cord, the paraplegic type. There are, however, numerous exceptions to this rule; not infrequently only one limb is paralyzed as the result of central diseases, and in very rare instances only a few muscles are involved. But in such cases there are usually other concomitant symptoms which indicate the site of the lesion. It must also be remembered that affections

of the peripheral nerves sometimes, though rarely, give rise to widespread paralyses. Thus, compression of the cauda equina may cause paralysis of the lower limbs. In one case, which I shall report in full at a later period, the hemiplegia which was present was due, in my opinion, to an affection of the peripheral nerves. Finally, a large number of the nerves of the body may be subjected at the same time to some lesion capable of producing paralysis.

Another important difference between peripheral and central paralysis is the fact that the affected muscles rarely atrophy to any appreciable extent in the latter. Thus, patients suffering from cerebral hemiplegia may have lost entire control of the paralyzed muscles for years, yet the difference in the circumference of the healthy and paralyzed limbs will be so slight that it can scarcely be detected. The small difference which does exist is undoubtedly due in part to the wasting of subcutaneous adipose tissue in the paralyzed side. In peripheral paralysis, on the other hand, muscular wasting always occurs, and usually with considerable rapidity. The same phenomenon is observed in a few diseases of the medulla oblongata and spinal cord, which are, however, recognizable by other attendant symptoms, to which we shall revert at length in the section on diagnosis.

Central paralyses are less frequently attended with sensory disturbances than the peripheral forms. This is especially true of cerebral forms, because the fibres which conduct motion and sensation are situated at a greater distance from one another in the brain than they are in other parts of the nervous system. In the peripheral nerves the motor and sensory fibres are mingled indiscriminately with one another, so that any noxious influence acting upon one set of fibres must necessarily affect at the same time the other. It is nevertheless true that, as an almost invariable rule, the sensory fibres are less involved than the motor, and, when the paralysis is not well marked, it is not infrequent to find that no sensory disturbance whatever can be detected in the distribution of the affected nerve. It is very difficult to explain this peculiar phenomenon, but it is none the less true that not only are the sensory disturbances less marked than the motor, but that the former also disappear more rapidly than the latter. Perhaps the sensory fibres, as has been conjectured, represent a lower grade of development than the motor, and are therefore less susceptible to external influences.

Unlike central paralysis, the peripheral form is usually attended with vaso-motor and trophic disturbances. This is readily understood when we remember that the peripheral nerves contain vaso-motor and trophic as well as motor and sensory fibres, and that the former must therefore be implicated to a certain extent in any affection of the nerves. These disorders are rarely present in cerebral diseases, but in one class of spinal affections they take a prominent part, which will form the subject of further comment at a later period.

Finally, the reactions of the affected nerves and muscles to faradism and galvanism differ markedly in peripheral paralysis from those observed in the vast majority of central nervous affections, as we show shall at length in the course of this section.

Peripheral paralysis may be due to various causes, such as traumatism, inflammation, rheumatic influences, etc., and as the clinical history varies considerably according to the cause, we shall enter into the symptomatology of each of these varieties in detail.

Injuries of Nerves.

We shall first consider that form of paralysis which is due to complete section of the nerves, the history of which has been very thoroughly investigated by means of physiological experiments, as well as by observations upon the human subject. This variety is produced by direct wounds of the nerve, such as arises from incised wounds with a knife, sabre, piece of glass, bullets, etc.

When a nerve has been cut across, a change in the white substance of Schwann first becomes evident. Within a very few days this portion coagulates and then breaks up into large, irregular masses presenting a double contour. These masses gradually disintegrate into smaller and smaller fragments, until finally nothing remains visible but an accumulation of fine fat globules which only present a single outline (the fat granules take up a greater space than the original medullary substance, and the nerve-sheath or neurilemma therefore appears swollen). After a few weeks these products of degeneration begin to be absorbed, and finally disappear in great part. The axis cylinder does not change so rapidly as the medullary substance, and some observers even maintain that it remains entirely unaffected. The weight of evidence, however, is to the effect that the axis cylinder also slowly undergoes a similar fatty degeneration, and is finally absorbed, so that at length the neurilemma is almost empty or encloses a small amount of the products of degeneration. The neurilemma also undergoes changes; it becomes thickened, its nuclei increase in size and number, and numerous white globules pass between it and the adjacent ones. The latter become converted into spindle-shaped cells, and finally into connective tissue, which is most abundant in the neighborhood of the sheath of the nerve; the nerve, therefore, undergoes a sclerotic change.

But even if a considerable portion of the nerve is excised, the cut ends may be again brought into union with one another by means of new-formed fibres. The manner in which this process is effected is still involved in obscurity. We know, however, from clinical experience, that such union will occur despite seemingly impassable obstacles, so that it is doubtful whether regeneration will not occur even when the cut ends of the nerves have been carried past one another, and thus project in opposite directions. This fact often proves a great obstacle to our therapeutic measures, as in cases of neuralgia in which, after excision of a portion of the nerve, we are anxious to avoid reunion of the cut ends.

The muscles supplied by the divided nerve also undergo important changes. The muscular fibrillæ diminish considerably in size, and the transverse striæ are not so well marked as in the normal condition; the fibres present a cloudy and even granular appearance. Increase of the interstitial tissue occurs as it does in the nerves; a large number of white blood globules appear between the muscular fibres, become converted into spindle cells, and finally into fibrous tissue. When these changes have not attained any considerable intensity they may entirely disappear, and all the parts involved (the nerves and muscles) resume their former normal appearance.

The central portion of the nerve may also undergo anatomical changes, though these are not constant. They consist of the development of neuromata or of an interstitial neuritis, which is usually limited to a small portion of the nerve immediately adjacent to the seat of injury. In some

cases, as we shall see at a later period, the neuritis may gradually spread along the nerve *per continuitatem*, and finally involve the membranes of the spinal cord, or the spinal cord itself, in the inflammatory process. This, however, rarely occurs when the nerve has been entirely divided.

In those cases in which the nerve has not been entirely divided, but has undergone slighter grades of injury, such as partial division, compression or contusion, the appearances presented by the organs involved are similar to those described above, but of less intensity.

The symptoms produced by complete section of a nerve depend upon the character of the latter, whether motor, sensory, or mixed. In all cases the trophic and vaso-motor fibres are paralyzed at the same time as the other fibres. The muscles supplied by the nerve are immediately and completely paralyzed, and remain so until regeneration has occurred. Sensation is not always entirely lost in the parts supplied by the nerve. Richet mentions a case of division of the median nerve which was not followed by the slightest loss of sensation. A considerable number of other cases have been reported, in which the lost sensation reappeared at a time at which regeneration of the cut nerve-fibres (they never unite by first intention) could not possibly have occurred. Arloing and Tripier have explained these cases by the existence of recurrent sensory fibres, which sometimes pass (especially at the periphery) from the trunk of one nerve to that of another.

The paralysis of the vaso-motor fibres is manifested by dilatation of the vessels and rise of temperature in the parts to which the fibres are distributed. After a variable period the temperature of the parts diminishes, on account of the ensuing atrophy and loss of mobility. In complete section of the nerves, the paralysis of the trophic fibres produces, as a rule, simple atrophy of the muscular tissue. In a few exceptional cases, however, other trophic changes have also developed.

When the division of the nerve has been incomplete, or when the injury is due to compression, contusion, or other source of irritation, the symptoms vary somewhat from those described above. The paralysis, in such instances, is not so complete, its distribution and severity depending upon the extent of the nerve injury; twitchings and contractures of the affected muscles may also be present. The sensory symptoms are also more complex, and consist of anæsthesia (which may not be diffused over the entire distribution of the affected nerve, but is sometimes interspersed with spots of hyperæsthesia); shooting pains in the peripheral distribution of the nerve, which sometimes assume a neuralgic character; a sensation of numbness and tingling in the parts. The trophic symptoms may be very prominent, and are similar in character to those described on page 94. In these cases, also, the symptoms of ascending neuritis are sometimes mingled with those due to the injury itself, but we shall defer their discussion until a later period. In order to give a clear idea of the multiplicity of the symptoms which may follow nerve injuries, we republish the following case from Weir Mitchell's treatise:

CASE I.—"*Wound of median and ulnar nerves; atrophy and contraction of flexor muscles; atrophy of all the hand muscles; neuro-traumatic arthritis; loss of sensation; moderate improvement; discharge.* G. L., aged thirty-one; lumberman; enlisted, May, 1861, Company C, 1st Minnesota Infantry. He was healthy to the date of the wound, which was received July 3, 1863. While advancing at a walk, and capping his *gun, a ball* entered the right biceps, three and a quarter inches above the

level of the internal condyle, and made its exit three and a quarter inches directly below the same condyle, wounding the main artery and the ulnar and median nerves. The hand and forearm flexed spasmodically, and the man, tying a handkerchief around the arm to check the flow of blood, walked to the rear, suffering with some pain down the front of the arm, but not in the hand. Motion and feeling were both absent. Three hours later the artery was tied. A cerate dressing was applied, and no splint was at any time used. The wound healed in two months, and at this time sensation and motion began to improve. He was admitted to Turner's Lane Hospital, December 24, 1863, when his case was thus described: The right hand is congested and a little swollen. In the flexor carpi radialis the loss from atrophy is one-half. In the other flexors, supinators and pronators the loss is one-third. In the thumb muscles proper and abductor minimi digiti it is one-fourth.

"The deep and superficial flexors and the thumb muscles are slightly contracted. Up to the third month the fingers were straight, but about that time they began to bend, and on admission were semi-flexed.

"The biceps acts very little; the supinator longus, although enfeebled, being competent to flex the forearm. Extensión is incomplete from partial contraction of the biceps, owing to the prolonged flexion of the arm; pronation is incomplete, but the supinating power is entire. The hand can be raised only to the arm level, when the contractions of muscles and the state of the wrist-joint check it. The thumb has one-third flexion and extension, owing to want of power.

"The first joints of the fingers are in good order; the second a little swollen and stiff; the third joints are rigid, enlarged and painful, especially in the index finger. The first joints have pretty fair mobility; the second but little; the third none at all. As the fingers are semi-flexed, the will can still act on the first phalanges, and slightly on the second, but the third rest bent and motionless. The same statement may be made as to the power to voluntarily extend them. The contracted state of the common flexors has now lessened, but the joint inflammations, which arose early in the case, have fixed the fingers in the vicious positions into which they were drawn by the muscular shortening.

"Touch is good in the arm; absent in the palm and palmar face of the fingers, except as to the thenar eminence. It is good on the dorsum of the hand, but defective on the back of the last phalanx of the first finger, the second and third phalanges of the second and third fingers, and on the metacarpal bone of the fourth finger.

"Electro-muscular contractility is lessened in the supinator longus; absent in the common flexors, though these have some voluntary power; nearly absent in the thumb muscles, the muscles of the fourth finger, and interosseal groups.

"The marked feature of this case was the obstinate and painful inflammation, and stiffness of the third joints of the fingers, and of the articulation of the wrist; yet these conditions were nearly entirely relieved by three months of passive motion, electrization by induced currents, and the dry wire brush, douches, and attention to the general health."

The changes in the electrical reactions of the affected nerves and muscles are extremely interesting and important, both from a diagnostic and prognostic point of view.

As a matter of course, no stimulation of the nerve above the site of division will be able to produce a response in the muscles to which the

nerve is distributed. The reappearance of muscular contractions after such stimulation is the best evidence which we possess that the divided ends of the nerve have become united.

Within a few days after the receipt of the injury, the peripheral portion of the *nerve* begins to lose its excitability to both the faradic and galvanic currents, and this keeps on steadily increasing until finally it is altogether lost, *i. e.*, the muscles will no longer respond to electrical stimulation of the nerve supplying them. Entire abolition usually occurs within a period of about two weeks (exceptionally, the excitability of the nerve to both currents is slightly increased for a few days after the injury, but after this time it always pursues the course described). As recovery occurs the irritability of the nerve to both currents slowly reappears and increases until it has resumed the normal proportions. It is a curious fact—and one which I have noticed on frequent occasions—that even after the paralysis is on the high road to recovery the nerve will not respond to electrical stimulus, although it readily allows the transmission of the impulse of the will, *i. e.*, the patient can move the muscles voluntarily, although they will not contract upon passing an electrical current through the nerve.

The *muscles* present different reactions according to the current employed. Their faradic excitability begins to diminish in two or three days, and then steadily decreases until it is entirely abolished within two to three weeks. In those cases which never recover it remains permanently absent; in those which are susceptible of improvement the musculo-faradic excitability reappears when recovery begins, and grows stronger in proportion as improvement progresses. The galvanic excitability of the muscles pursues an entirely different course. For the first week or ten days it is either entirely unaffected, or is perhaps slightly diminished. But at this time the galvanic excitability begins to grow stronger and increases in proportion as the degeneration of the muscles occurs, until finally the paralyzed muscles react to a much milder current than the corresponding ones on the unaffected side of the body.[1] The increase in the galvano-muscular excitability is sometimes so great that I have frequently observed contraction of the paralyzed muscles from a current of two cells, whereas the healthy muscles would only respond to ten cells. As a rule the character of the muscular contractions is somewhat different from that of healthy muscles—they appear at a longer period after the passage of the current, and continue for a longer time. This change in the muscular excitability is known as the degeneration-reaction (*entartungs-reaction* of Erb). If no improvement occurs in the condition of the muscles the galvanic excitability begins to diminish after a variable period, and finally becomes entirely extinct. As a rule recovery is impossible after this has occurred, but some exceptions have been reported, and two such have come under my notice. When improvement does take place the increased galvanic excitability gradually diminishes until finally it becomes normal.

---

[1] There are also other more complex changes in the reactions of the muscles to galvanism. In healthy muscles the reaction at the closure of the negative pole is much feebler than that at the positive; in the paralyzed muscles the former reaction increases much more rapidly than the latter, so that it finally becomes more powerful. Furthermore, the reaction at the opening of the negative pole is greater than that at the opening of the positive pole. In the paralyzed muscles the latter increases much more rapidly than the former, and finally become superior to it. This has been termed by Brenner "reversal of the formula."

The remarks just made only hold good for complete division of the nerve; when the injury has been less severe, the changes in the electrical reactions are not so well marked.

In the mildest forms the nerves as well as the muscles present entirely normal reactions to both currents, and these cases always recover very rapidly. There are, however, numerous intermediate groups between this class and the variety described above, and we find that the electrical reactions vary according to the severity of the nerve-wound. In some cases there is simple diminution of nerve and muscular excitability to both currents, a certain grade of irritability being preserved throughout the entire course of the affection. In others the musculo-faradic excitability is lost, while the musculo-galvanic excitability, though increased, does not present the "reversal of the formula" to which we have referred. Numerous other variations have been reported, but it is unnecessary to enter into their discussion; they are merely the result of variable degrees of change in the anatomical condition of the affected parts.

Acute Neuritis.

The consideration of peripheral paralysis from inflammation of the nerve, or neuritis, next claims our attention. Neuritis includes two varieties, viz., the acute and chronic.

Acute neuritis is usually the result of injury of the nerve, either in gunshot or other wounds; idiopathic cases appear to be exceedingly rare. As a rule the inflammation only affects the injured nerve or nerves, or, in idiopathic cases, is limited to one nerve, or perhaps to a plexus. In extremely rare instances, however, as in the cases recently reported by Eichhorst[1] and Leyden,[2] a very large number of nerves situated in different parts of the body are simultaneously involved.

In acute neuritis the nerve appears swollen, and is usually of a speckled reddish color; small punctate hemorrhages are visible, scattered here and there throughout the sheath and tissue of the nerve. The capillaries are enlarged and are surrounded by numerous leucocytes which have also escaped between the nerve-fibres; the white substance of Schwann has usually undergone fatty degeneration and has been removed in places, so that the sheath of Schwann collapses. The axis cylinders present variable appearances; sometimes they are in a condition of simple atrophy, sometimes cloudy or granular; a certain number disappear entirely.

The disease usually begins with a chill and considerable fever; intense pain at once develops along the course of the affected nerve, which becomes swollen and exquisitely tender to the touch, so that the slightest contact produces excruciating agony; in some cases a red livid streak is visible over the nerve, at other times the integument is œdematous. These symptoms may be combined, in the beginning, with delirium. The patient suffers continuously from pain *in loco morbi*, and this is accompanied at times by sharp, shooting pains in the distribution of the nerve; paralysis of the muscles which it supplies rapidly supervenes. The skin is very hyperæsthetic, but this condition may change

---

[1] Virch. Arch. Bd. LXIX. [2] Charité-Annalen, Berlin, 1880.

to anæsthesia after a few days; the reflex excitability of the parts is lost. The affected limb is kept motionless on account of the pain, and contracture of the muscles rapidly occurs in some cases. After a variable period the inflammation may undergo resolution, and the symptoms then subside, or it may pass into a chronic condition. The following case, reported by Weir Mitchell, is an excellent example of the latter variety:

"J. C., sergeant, consulted me on account of loss of power in the arm, with severe neuralgia. At Gettysburg he received a ball-wound in the left neck, splintering the clavicle and emerging through the trapezius. Some fragments of bone were lifted out of the wound, which did well until a week later, when, on the way to Washington, he was suddenly taken with a chill of some severity, followed by high fever. At the same time the whole arm began to ache, darting pains shot up and down it, and the skin on the inside of the arm, below the axilla, was seen to be red. The nerve tracks were extremely tender. On the third day the whole arm was somewhat swollen, and the darting and aching pain was only subdued by frequent hypodermic injections. His first notable relief was obtained by an application of cut-cups to the neck and shoulder, and gradually the pain lessened to its present grade of severity. The ulnar and median were hard, enormously enlarged, and very tender. J. C. described himself as having been made delirious by the earlier pain of his disease; and even when seen by me after it had been abated, he showed very plainly that the mind as well as the body had suffered—his memory being impaired, and his temper excessively irritable."

As we have stated above, two very interesting cases have been reported in which the neuritis involved a large number of nerves in various parts of the body. The following is an abstract of Eichhorst's case which he described under the term acute progressive neuritis:

"A woman, who was apparently suffering from quotidian intermittent fever, was suddenly paralyzed in the left peroneal nerve, which lost faradic excitability within twenty-four hours. The other peripheral nerves gradually became paralyzed, so that all the limbs were paralyzed in ten days. Amblyopia then developed and finally death.

"Upon the autopsy no change was found in the brain or cord; the peripheral nerves, however, were all of a dirty grayish red color. The perineurium was very rich in vessels, which were surrounded by lymphoid cells; the blood-vessels were thickened in places. The nerve-fibres were only degenerated in the neighborhood of the perineurium."

Leyden's case, which has been referred to above, is somewhat similar to this in its general outlines; it also began suddenly with acute symptoms, but the neuritis afterward passed into a chronic stage. The patient died a year later from uræmia, the result of small contracted kidneys.

As we shall see further on, these exceptional cases bear very interest- in grelations to the pathology of certain diseases of the spinal cord.

Chronic Neuritis.

Chronic neuritis is sometimes an outcome of the acute form of the disease. It is also due to direct injury of the nerves, to compression in consequence of various lesions in their vicinity, such as the pressure of tumors, excessive callus, etc., the spread of inflammation from adjacent *parts, exposure, etc.* An attempt has been made to divide the affection

into two forms, viz., perineuritis or inflammation of the sheath of the nerve, and neuritis proper or inflammation of the nerve itself. Such a distinction is impracticable, however, from a clinical point of view, and very often from an anatomo-pathological standpoint, since both these lesions are often combined.

When the inflammation is more interstitial in character the sheath of the nerve and the interstitial connective tissue are thickened from an increase of spindle cells and new formed fibrous tissue; in consequence of this sclerotic change the nerve-fibres are pressed upon and undergo atrophy; adipose cells sometimes develop between the fibres. In parenchymatous neuritis the nerve-fibres undergo the most prominent change. The axis cylinders are frequently increased in size, though sometimes they are atrophied; the white substance of Schwann has degenerated in some places and disappeared in others, allowing the sheath to collapse. The blood-vessels are thickened, and may be surrounded by capillary extravasations of blood.

The muscles supplied by the affected nerve present the same changes as those described on page 165, after division of a nerve.

The symptomatology varies considerably, according to the intensity of the inflammatory process. The disease usually begins by a feeling of numbness and weakness in the distribution of the nerve involved. After a longer or shorter period the patient begins to suffer from pain. In the beginning this consists of mere soreness along the course of the nerve, which soon grows more severe, until finally an intense steady pain is felt. In some cases paroxysms of shooting pain are experienced at longer or shorter intervals; their character is very similar to that of attacks of neuralgia, but the former present a greater tendency to propagation in a centripetal as well as a centrifugal direction. The nerve is always tender on pressure (when accessible to the touch), but no circumscribed painful spots are observed, such as we find in true neuralgia. When the nerve is situated directly underneath the skin it is often found to be enlarged. Mistakes are readily made, however, in this respect, and we should therefore always compare the inflamed nerve with the corresponding one on the opposite side of the body. When the inflammatory process is a severe one the integument supplied by the affected nerve is profoundly anæsthetic; in less marked cases spots of superficial anæsthesia may alternate with patches of hyperæsthesia. The patients also complain of sensations of numbness and tingling or a feeling of formication. The muscles undergo a greater or less amount of atrophy, according to the degree of implication of the nerve-fibres. Fibrillary twitchings and spasms are often noticeable in the paralyzed and paretic muscles; contracture of these muscles may also take place.

The electrical reactions present marked variations. Thus the affected nerve, in slight cases, may be more excitable to both the faradic and galvanic currents than in the normal condition. As the nerve-changes grow more intense, the excitability to both currents diminishes, and finally disappears altogether. Similar variations are noticeable in the electrical muscular reactions. Those muscles which have undergone profound atrophy always present the degeneration reaction (vide p. 168), while those least affected may react normally to both currents. Erb's so-called "middle forms" of degeneration reaction may also be present. It is not at all infrequent, especially when several nerves are involved, to find all these different varieties of electro-muscular reactions in one individual.

been a strong tendency among neurologists to entirely discard the old doctrine of reflex paralysis, but there is no doubt that in a few cases the theory of reflex inhibitory action is alone sufficient to account for the paralysis produced. Thus Landry reports a case of paralysis, associated with anteversion of the uterus, which disappeared immediately after the organ was replaced in its normal position. M. Rosenthal observed the disappearance of a suddenly-developed paraparesis after the extraction of a needle which had been introduced into the vagina. Madge reports a case of paralysis developing during pregnancy, which disappeared after the delivery of a dead four months fœtus. Fuller [1] mentions the case of a boy, æt. 3 years, suffering from paralysis of the right arm and of both legs, which was relieved by the expulsion of fifty-three lumbricoid worms. The theory of reflex inhibition must also be invoked to explain those cases (of which Mitchell has reported several), in which an injury to one nerve has produced paralysis in the distribution of another, and in such a short period of time that no anatomical lesion could have been produced.

But this theory will evidently not suffice for all cases of this nature. In many instances post-mortem examination revealed the presence of inflammatory processes in the spinal cord or its meninges, in cases which had been regarded during life as examples of reflex paralysis from bladder or uterine disease, etc. The histories of the older cases of this character are deficient in microscopical examination of the medullary tissue, and must therefore be discarded. In order to explain the cases included in this category in which anatomical lesions were found in the spinal cord, resort was had to the theory of neuritis migrans, and this doctrine has been favorably entertained by the majority of living pathologists. In addition to the clinical aspects of the question, physiological experiments were also adduced to substantiate it. Thus, Tiesler, Klemm, Feinberg, and Niedick made numerous experiments upon animals by producing irritation of the sciatic nerve in some portion of its course, either by injecting a foreign matter into its substance, or by cauterizing it with nitrate of silver or caustic potassa. With the exception of Feinberg, these observers announced as the results of their investigations that the irritation of the nerve in the manner referred to produces neuritis at the irritated spot, and that, furthermore, evidences of inflammation appear in the course of the nerves, although the nerve-tissue may be perfectly healthy between two inflammatory foci. In addition, scattered foyers of myelitis were found disseminated throughout the cord. None of these experimenters, however, resorted to microscopical examination of the tissues, nor did they compare the appearances presented with those found in healthy animals. Feinberg found, as the result of his investigations, that neuritis was produced at that portion of the nerve which had been irritated, but that the more central parts of the nerve were intact. He nevertheless obtained evidences of myelitis in the cord. This observer is inclined to regard the myelitic process as due to reflex irritation of vasomotor nerves (contraction and secondary dilatation of the medullary vessels).

In December, 1877, Ottomar Rosenbach published an article in Kleb's "Archiv. f. exp. Path. u. Pharmak.," in which he arrived at entirely opposite results from those of the observers previously mentioned. Dr. Rosenbach made a series of very careful experiments upon the pneumo-

[1] The Lancet, December, 1865.

gastric and sciatic nerves in rabbits, and, although he could develop a perineuritis at the irritated point, in not a single instance was he able to discover any evidences of neuritis migrans or of secondary myelitis. All his experiments were accompanied by careful microscopical examinations—a precaution which had been omitted in the above-mentioned researches. Rosenbach also calls attention to the fact that no controlling observations were made upon healthy animals by either Klemm, Tiesler, Feinberg or Niedick, and that many of the appearances which the latter regarded as pathological were, in fact, perfectly normal.

Similar experiments have been made more recently by H. Treub [1] and have entirely substantiated the conclusions arrived at by Rosenbach.

Whether or not the experiments of Rosenbach and Treub disprove the possibility of the production of neuritis migrans, we shall not discuss now. We are, however, warranted in the assertion that its existence has not been experimentally established, and that other and more careful experiments are necessary to settle this vexed question.

We must therefore rely for a solution of the problem upon pathological and clinical data.

Leyden [2] reports two cases in both of which the paralyses were secondary to disease of the bladder, and in which the autopsy showed the existence of widespread myelitic softening. The myelitis started from that part of the cord in which the nerves supplying the bladder originate, and we are therefore naturally led to suppose that the inflammatory process passed upward from the bladder, and along the nerves, until it reached the cord. But Leyden himself remarks that there is no positive proof in support of this hypothesis. I have been unable to discover any other analogous cases of equal importance after a survey of the medical literature which has appeared since the publication of Leyden's work.

The case of Wm. W., which I have reported above, appears to me to fill an hiatus in this direction. The primary affection was evidently a neuritis of the internal cutaneous, external cutaneous, and ulnar nerves, caused by an extension of inflammation from the boil situated on the elbow. The fact that the boil was primarily seated over the olecranon process, and from thence spread internally and externally, is a sufficient anatomical explanation of the fact that the three nerves in question were implicated to the exclusion of the other nerves situated in the fold of the elbow. At a later period pain and tenderness became evident along the course of the ulnar nerve in the arm, and the nerve could be traced as a thickened, indurated cord from the back of the inner condyle, along the inner side of the arm into the axillary space. These phenomena undoubtedly indicated extension of the inflammation along the ulnar nerve. At a still later period tenderness became evident along the course of the brachial plexus in the neck, indicating the further extension of the neuritis along the nerve-trunk.

The next nerve to become involved was the auricularis magnus, as was evidenced by the appearance of anæsthesia and of the trophic eruption referred to previously, in its distribution to the lobe of the ear and to the side of the neck. Now the ulnar nerve which had been previously implicated arises from the eighth cervical and first dorsal nerves, while the auricularis magnus is given off from the second or third cervical nerves. If we acknowledge that the implication of the auricularis magnus was sec-

[1] Arch f. exp. Path. u. Pharm., p. 398, 1879.
[2] Klinik f. Rueckenmarkskrankheiten.

ondary to that of the ulnar nerve (and no other explanation is open to us), we are forced to conclude that the affection of the former was caused by some inflammatory process within the spinal canal. This idea is still further strengthened by the subsequent appearance of similar spots of anæsthesia and of the trophic eruption in other portions of the body (deltoid muscle, scapula, thigh). What the nature of the medullary lesion was we are unable to state. It may have been a disseminated chronic myelitis, or the inflammation may have been limited to the meninges. The spinal symptoms were so slight that it would be rash to venture a differential diagnosis between these two conditions.

It appears to me that this case demonstrates from a clinical standpoint (and with almost as much positiveness as a successful physiological experiment) that ascending neuritis is capable of developing secondary inflammatory changes in the cord, by means of a simple extension of the neuritic process *per continuitatem*.

This tendency to the upward spread of the chronic inflammatory process along the nerve which was primarily affected, is quite commonly met with in cases which belong to the category at present under discussion. As a rule, however, the spread of the inflammation stops at the nerve plexuses, and does not continue as far as the spinal cord.

There is also strong reason to believe that neuritis may be propagated downward along the course of the nerve, as appears to have been the case in the following example : A patient fell upon the left shoulder, and this injury was followed by symptoms of neuritis in the circumflex nerve. Within a month pain and tenderness were felt in the brachial plexus, with shooting pains along the circumflex nerve and very marked atrophy of the deltoid muscle. This was followed by the development of pain and tenderness along the nerve-trunks in the arm, and shooting pains along the distribution of the ulnar nerve in the forearm.

The development of pain and tenderness in the nerves of the arm subsequently to the appearance of these symptoms in the course of the brachial plexus probably indicated a downward spread of the neuritis.

The results of autopsical examination have also shown the actual existence of neuritis descendens.

Rheumatic Paralysis.

Another large category of peripheral paralyses is that which is due to so-called rheumatic or atmospheric influences, and is known as rheumatic paralysis (also called paralysis *a frigore*). Our knowledge of the lesions which are produced in this form of the disease is purely hypothetical, so far as regards the milder varieties, which never prove fatal. The paralysis was formerly supposed to be reflex, but there is good reason to believe that this view is incorrect. Thus, Frerichs reports a case in which a child, æt. 3 years, became paraplegic after having sat upon a cold stone for several hours. The case terminated fatally, and the autopsy showed the presence of exudative meningitis throughout the entire spinal canal. From certain of the symptoms in the peripheral forms of rheumatic paralysis, it is supposed that the affection is due to various grades of neuritis or perineuritis, which may sometimes be so severe as to lead to degeneration of the nerves and muscles, such as we have described as the result of complete division of the nerve. The modus operandi, however, of the production of such lesions is as little known as is that of the de-

velopment of bronchitis after exposure, etc. Rheumatic paralysis develops either as a consequence of cold or from exposure to draughts, especially when the patient is perspiring and is not exercising at the time. In a large proportion of cases the facial and ocular muscles are involved, and as we shall see at a later period, the paralysis may vary greatly in intensity; some attacks recover spontaneously in a few days, others are incurable. Not infrequently rheumatic paralysis occurs during sleep, when the patient is lying in a draught. It may develop within a few minutes after the operation of the exciting cause, or not until the lapse of a couple of days. It is usually unattended with pain or other sensory disturbances, and the duration of the disease generally varies according to the electrical reactions. It was in this form of paralysis that the "degeneration-reaction" was first observed by Baierlacher. In the most severe cases the degeneration-reaction is as well marked as that described after the complete division of a nerve, and in some all electrical reactions of the nerves and muscles may be entirely lost. But as in paralysis from injuries of nerves, there are numerous gradations between these severe types and the slight forms in which the muscular and nervous electrical reactions are entirely normal, and complete recovery occurs within a few days.

The more intense forms of rheumatic paralysis are not infrequently followed by spasms and contractures of the affected muscles. The latter condition is very liable to lead to permanent deformity, which may seriously interfere with the progress of recovery.

Atrophy of the muscles does not appear to be as marked as in the varieties of paralysis which we have previously described.

Paralysis following Infectious Diseases.

The occurrence of paralysis as a sequence of various general diseases had been recognized by a considerable number of clinical observers for a long time, but it is only within the past fifteen years that especial attention has been called to these affections. They were at first regarded as purely functional, *i. e.*, no anatomical lesion was supposed to be present in these cases; but the pathological investigations of the last decade have shown that they are due, in the majority of instances, to well-defined lesions which are situated sometimes in the brain and spinal cord, sometimes in the peripheral nerves alone. But quite a number of cases have been reported very recently in which even the most careful microscopical examination failed to show the presence of any morbid changes. Déjerine gives the results of his examination of three cases of diphtheritic paralysis, in one of which the muscles of the arms and neck, in another both arms, and in the third all the limbs were affected. The morbid appearances were similar in all these instances, and were chiefly confined to the anterior roots of the spinal nerves. The nerve-sheaths and interstitial connective tissue showed a very marked increase of cellular elements, there was a similar increase in the nuclei of the neurilemma, and the white substance of Schwann had degenerated into a sort of granular mass; many of the axis cylinders had entirely disappeared.

Buhl found, in the same disease, that the roots of the nerves were thickened and had undergone yellow softening in places; the sheaths of the nerves and their interstitial connective tissue showed a cellular infiltration which Buhl regarded as identical with the diphtheritic infiltration in the mucous membrane of the pharynx.

Bernhardt describes the following appearances in a case of paralysis

of the radial nerve following typhoid fever: the capillaries were moderately distended with blood globules, and numerous capillary hemorrhages were noticed along the walls of the vessels; the axis cylinders had disappeared in great part, and the white substance of Schwann had undergone fatty and granular degeneration.

Similar changes have been noticed in the paralyses following dysentery, measles, etc. We may also remark that softening and disseminated myelitis have been found in the cord, and capillary hemorrhages, softening, etc., in the brain in cases of central paralysis following acute infectious diseases.

In not an inconsiderable number of autopsies, however, no changes have been discovered, notwithstanding the existence of well-marked paralysis (which may even have been the direct cause of death), and we are therefore forced to conclude that some of these forms may be due to the direct paralyzing action of the specific virus upon the nerve-tissues.

Clinical History.—Paralysis may occur as a sequel of diphtheria, dysentery, whooping-cough, variola, scarlatina, rubeola, typhoid fever, intermittent fever, pneumonia, acute articular rheumatism.

Some of these affections are much more frequently followed by paralysis than others, and diphtheria is by far the most important in this particular. We shall therefore devote our chief attention to a description of diphtheritic paralysis.

There does not appear to be any special relation between the severity of the primary disease and the frequency of occurrence of post-diphtheritic paralysis. All of my own cases have occurred after mild attacks of diphtheria, and it is not infrequent to find the paralysis develop, although the throat trouble was so slight that the case would have been called one of simple pharyngitis, were it not for the existence of undoubted cases of diphtheria in the immediate neighborhood.

The loss of motor power usually develops within the first four weeks after the beginning of the disease, although in rarer instances it may not occur until after the lapse of several months. In the beginning the muscles of the velum palati are generally involved; this is shown by the nasal character of the voice, and by the fact that the regurgitation of fluids, which may have been present in the early period of the disease, now reappears. Upon inspection the velum palati and uvula are found to have lost their tonicity, and perhaps one pillar of the fauces hangs lower than the other (we have not met with any reports of cases, however, in which only one pillar was paralyzed). Reflex action from the velum palati is lost, and the parts do not react even to the most energetic stimulation. The electrical reactions of the affected muscles vary considerably. In the milder cases they are unaffected, in others there is simple diminution both to the faradic and galvanic currents; a few instances have been reported in which the faradic excitability was abolished and the galvanic excitability markedly increased. The latter condition has been noticed by Leube, Krafft-Ebing, Rosenthal, and Joffroy, so that its occurrence is unquestioned.

In the majority of cases some of the muscular structures of the eye are the next to suffer. Within a few days after the development of the faucial disturbance the patients suffer from paralysis of the sphincter of the iris and the tensor muscle of the choroid. This is evidenced by dilatation of the pupil and loss of the power of accommodation, so that the patient can see distant objects distinctly, while vision for near objects is *markedly impaired.*

More rarely some of the other ocular muscles are also involved, especially the rectus internus and externus. Eulenburg states that he has sometimes noticed a rapid disappearance of the paralysis in one ocular muscle (in from twenty-four to forty-eight hours) and its sudden appearance in another.

Rosenthal reports the occurrence of facial paralysis following diphtheria. "The middle paralyzed muscles of the face had lost their faradic contractility, but preserved their irritability to the continuous current. Even after the disappearance of the paralysis, the same difference existed with regard to the action of the two currents."

Paralysis of the muscles of the limbs and trunk is not infrequently present. It is rarely observed except in cases in which the velum palati has been affected, and usually develops after the latter begins to improve or even after the faucial disorder has entirely disappeared. The paralysis may begin either in the upper or lower limbs, and gradually spreads to the rest of the body. The muscles of the neck and trunk are not very infrequently involved, and in such cases paralysis of the diaphragm is liable to occur. These forms present naturally a very gloomy prognosis.

The sensory disturbances are very slight, and usually limited to a feeling of numbness, with or without anæsthesia, in the paralyzed parts. The affected muscles are very apt to undergo atrophy, though this trophic change does not develop with any rapidity. In two of my cases loss of power in the lower limbs slowly developed, with considerable wasting of the muscles and some numbness and anæsthesia. In both of these cases incontinence of urine occurred, and the patients did not possess normal control over the rectum. Neither of these individuals had suffered from any faucial paralysis.

The electrical reactions of the affected muscles present great variations, and various forms of reactions may be observed in the same individual, according to the severity of the paralysis. In a case recently reported by Fritz,[1] the muscles of the calves of the legs only responded to a very strong faradic current, while the muscles of the ball of the thumb were entirely inexcitable to this current. Exploration with the galvanic current showed the presence of marked degeneration-reaction in the thumb muscles. As the paralysis of the legs increased, the degeneration-reaction also developed in the muscles of the calves.

As a rule, the prognosis of post-diphtheritic paralysis is very good. The patients usually recover within one to two months, especially when the affection is limited to the muscles of the velum palati or the eye.

The favorable prognosis, the occurrence of the degeneration-reaction, and the results of autopsical examination (vide p. 177), render it very probable that the majority of cases of post-diphtheritic paralysis, whether circumscribed in character or general, are due to an affection of the peripheral nerves (cellular infiltration, etc.). We must remember, however, that no lesions are discovered in a certain proportion of cases.

Typhoid fever ranks next in importance to diphtheria among infectious diseases as the cause of paralysis. Like diphtheria, it may be followed by paralysis in different parts of the body (ocular muscles, face, paraplegia, hemiplegia, various nerves). These paralyses develop, as a rule, as a sequence of the disease, but they have also been observed at the height of the affection. They differ from those forms following diphtheria in the fact that they usually run a much more protracted course. With this ex-

[1] Charité-Annalen, p. 255, 1880.

ception, their clinical history as regards atrophy of muscles, electrical reactions, etc., is exactly similar to that of the post-diphtheritic affection. Westphal [1] has recently reported a case of post-typhoid paralysis, which is so remarkable, in some respects, that we shall republish a portion of the history in full:

"The patient, æt. 30 years, recovered from an attack of typhoid fever with a paraparesis of the lower limbs. At the end of nine months she was unable to stand or walk; the peroneal muscles were completely paralyzed, the tibiali antici retained some power. No sensory disturbances, no muscular spasms; the bladder and rectum acted normally.

"The peronei longi and gastrocnemii muscles could be contracted voluntarily and reacted, though very feebly, to the faradic as well as to the galvanic currents.

"*Both tibiales antici muscles contracted under the influence of the will and of the faradic current, although the strongest galvanic current was unable to produce the slightest contraction.* (This remarkable condition is called isofaradic reaction by Westphal.) Under the influence of treatment with the faradic and galvanic currents, this peculiar phenomenon soon disappeared. The tibiales antici muscles gradually began to respond to the galvanic current, in about two months presented the degeneration-reaction, and later became normal."

Variola, rubeola, scarlatina, dysentery, cholera, erysipelas, pneumonia, and acute articular rheumatism are also followed by paralyses at times, which occur, however, much more infrequently than after diphtheria and typhoid fever. Their clinical history is in all respects similar to that of those described as occurring after the latter diseases, and we shall therefore refrain from entering into a description, which would be a mere recapitulation of the remarks previously made.

Numerous other nervous disturbances, such as acute ascending spinal paralysis, multiple cerebro-spinal sclerosis, locomotor ataxia, etc., have been observed as sequelæ of the diseases just mentioned, but their discussion is beyond the scope of this article.

Toxic Paralysis.

The next class of cases which require our consideration are the *toxic paralyses*, which occur in consequence of poisoning with lead, arsenic, etc. Lead is such an important factor in the production of paralytic and other nervous disorders that we will enter into the discussion somewhat in detail.

This metal may be introduced into the system in a thousand different ways, by inhalation, by the mouth or from external application to the skin. Inhalation may occur from living in newly-painted rooms, from working in factories in which lead is employed and in which the air is impregnated with the poisonous particles (as in white lead manufactories, in the operation known to painters as flatting, etc.), from taking snuff which has been wrapped in lead foil, etc. It may be swallowed in confectionery colored with lead salts, in articles which have been wrapped in lead foil, in ale drawn through lead pipes (especially that which is drawn early in the morning and has lain in the pipes over night), in water drawn from lead-lined cisterns, in bread, the flour composing which has been ground in

[1] Charité-Annalen, p. 376, 1880.

stones filled with lead, in articles cooked in vessels lined with a lead glaze, etc., etc. It is absorbed through the skin in various trades, such as plumbing, from handling type, using hair-dyes and cosmetic, etc.

Among 1,213 cases of chronic lead poisoning collected by Tanquerel des Planches, 406 occurred in white lead manufacturers, 305 in house painters, 68 in color grinders, 63 in red lead manufacturers, 54 in earthen pottery manufacturers, 52 in type-founders, 47 in carriage painters, 35 in lapidaries, and 33 in ornamental painters. It is sometimes extremely difficult to determine the source of the entrance of the lead into the system in individual cases, and in several instances we have been unable to discover its origin although the most indubitable evidences of lead poisoning were present.

The habits of the patient should be carefully inquired into, and his surroundings examined. Negative statements with regard to the impossibility of the entrance of lead into the economy should never be accepted when well-marked symptoms of poisoning are present; the physician should always satisfy himself personally of the condition of affairs. Children are very apt to put into the mouth any substance with which they come in contact, and it is therefore advisable, in cases of lead poisoning in children, to carefully examine their toys, etc. The determination of the mode of entrance of the poison into the system is, of course, of the first importance, as we cannot expect any improvement in the symptoms until its further admission has ceased.

Lead palsy rarely occurs without any previous symptoms of poisoning. The patients have usually suffered for a long time from obstinate constipation, the appetite is poor, the skin assumes a peculiar ashen-gray appearance, they complain of a metallic taste in the mouth, the tongue is habitually furred, and the expired air exhales a disagreeable, sickening odor. The pulse is usually slow and hard, the surface of the body is dry, the gums present the blue line. As a rule, the patients have suffered, prior to the paralysis, from one or more attacks of lead colic. Lead paralysis may not occur until the above-mentioned symptoms have lasted for years, in other cases it may develop after a very short exposure to the deleterious influence of the poison. In one case under my own observation the paralysis began to develop within a month after the patient had begun work in a white lead factory, although prior to that time he had been in perfect health and had never, so far as I could ascertain, been exposed to the influence of lead.

Lead palsy is usually bilateral, but in some cases it may be limited to only one side. In the majority of instances the extensor muscles of the forearm are the only ones affected in the beginning of the malady, though other groups of muscles may be involved at a later period. Before the disease develops, the patients usually suffer from tremor of the arms on exertion, and often from neuralgiform pains and anæsthesia. The weakness of the muscles generally develops gradually and increases until the production of paralysis. Several months may elapse between the time at which the patients first experience weakness of the limb and the development of complete paralysis. In exceptional cases, however, the loss of power develops quite suddenly. Thus the patient may retire to bed, feeling entirely well, or perhaps complaining of numbness in the arms, and upon waking in the morning, the palsy may be complete or almost so. As we have previously stated, the extensor muscles of the forearm are the ones usually involved, giving rise to the characteristic deformity known as wrist-drop (when the forearm is held horizontal in complete pronation,

the hand drops down almost at right angles to the arm, and in the severest cases not the slightest degree of extension can be performed). It is a remarkable fact, and one which is extremely important from a diagnostic point of view, that the supinator longus and brevis remain unaffected by the disease, though these muscles, as well as the extensors, are supplied by the radial nerve.

Lead palsy is usually accompanied by rapid atrophy of the affected muscles so that the appearances presented are very characteristic. Upon examining the forearm the normal rotundity of its posterior surface is found to be lost, and the atrophy is sometimes so extreme that the finger can be pressed into the interosseous space; immediately adjacent, however, the prominent supinator longus is seen passing down the outside of the forearm in its natural proportions. Although the freedom of the supinator longus from paralysis and atrophy is almost always observed, there are some exceptions to this rule, and I have myself observed two cases in which this muscle was involved to the same extent as the extensors. The electrical reactions of the affected muscles vary considerably. In some cases there is merely a diminution of their faradic excitability, in others their galvanic excitability also diminishes. These reactions may continue throughout the entire course of the disease. The degeneration-reaction is also observed in some cases, and, it is said, always in those forms which are attended with very rapid muscular atrophy. I have at present, however, a case under observation in which the atrophy is so great that the finger can be pressed into the interosseous space, but in which the faradic excitability of the muscles, although not very distinct, is entirely proportionate to the small amount of muscular fibre left intact; the galvanic excitability is not increased. A number of cases of this kind have come under my notice. Eulenburg has shown that increased mechanical and reflex excitability of the muscles may be present in cases in which their galvanic contractility is increased.

It has been stated by numerous writers that in cases of wrist-drop the flexors of the arm are also partially paralyzed. This statement is due to a mistake in observation, as can be readily shown. If the hand is flexed upon the forearm, actual measurement will show that the flexor muscles are $1\frac{1}{4}$ to $1\frac{1}{2}$ inches shorter than when the hand is extended (this is readily determined by making a mark in the bend of the elbow and measuring from it to the tips of the fingers while flexed and extended). But as muscular power is due to contraction of the muscles, the power of the flexors in wrist-drop must necessarily be markedly diminished, since the mere position in this affection causes a shortening, as we have seen, of $1\frac{1}{4}$ to $1\frac{1}{2}$ inches. If the paralyzed hand is held by the observer in a position of moderate extension and the patient be then directed to exert the flexors, their power will be found undiminished.

In exceptional cases the paralysis does not begin in the extensors of the forearm. In two of my patients the loss of power began in both deltoids, and had progressed to such an extent that these muscles had almost completely wasted away before the extensors began to be affected. In another case I observed paralysis of the extensors of the feet before those of the arms became implicated. In a not very inconsiderable number of cases the paralysis begins in the usual situation, but then gradually spreads to other muscles of the body, so that the disease may run the course of a progressive muscular atrophy. Buzzard[1] mentions an inter-

[1] Brain, p. 121, vol. i.

esting case in which the right hand and the left foot were paralyzed; the supinators were not affected. The faradic excitability of the extensors was lost in the affected parts, and diminished in the left hand and right foot; there was increased excitability to galvanism; no blue line on the gums. The diagnosis was chiefly based on the electrical reactions and the freedom of the supinators from paralysis. Careful examination showed that the cistern from which the drinking-water was drawn contained a roll of lead pipe which had been left there while repairs were being made.

The pathology of lead-paralysis is still very obscure. In cases of chronic lead-poisoning, lead has been found in the brain, cord, muscles, bones, etc., a larger quantity being found in the spinal cord than in the muscles. Hitzig was of the opinion that the paralysis was due to the direct deposit of the metal in the affected muscles, but this view has been successfully combated by Bernhardt, who found that the unaffected supinator longus contained proportionally almost the same quantity of lead as the atrophied extensor muscles.

The anatomical changes in this disease have not been very thoroughly investigated. Lancereaux reports one case in which the cervical enlargement of the spinal cord presented a soft consistency and was not as large as normally. Some of the nerve-roots springing from this portion of the cord were atrophied, and the nerve-fibres had undergone granular degeneration. Vulpian[1] found, in a case of lead-paralysis, marked poliomyelitis with colloid degeneration and atrophy of some of the ganglion cells, an increase in the number of nuclei, and patches of sclerosis in the roots of the cervical enlargement of the spinal cord. C. V. Monakow,[2] who made an extremely careful examination, corroborates Vulpian's observation in great part. Westphal, Gombault, and Friedlander, on the other hand, found no changes in the spinal cord. Various lesions have been observed in the radial nerve (granular and fatty degeneration, increase of nuclei, etc.), but no definite results have been obtained, and it is undecided whether these changes are primary or secondary. Mayor[3] found the following changes in the intramuscular nerves of the extensor communis pollicis in a case of lead-paralysis: the myeline in rounded drops; disappearance of the axis cylinders; nuclei of some of the fibres more numerous, with complete disappearance of the myeline; the sheath of Schwann persistent; a large number of the nerve-fibres entirely healthy.

The paralyzed muscles have been found paler than normal, the transverse striæ not well marked, and the nuclei of the sarcolemma increased in numbers; there is increase of the interstitial tissue, and a new development of adipose tissue.

It is very evident from these manifold changes that pathological anatomy has not yet determined the real nature of the disease. Hitzig's theory of a local affection of the muscles has been previously referred to, and the weak basis upon which it is founded, pointed out.

The question as to the peripheral or spinal character of the paralysis is not definitely settled, though there is a strong tendency at present to regard it in the light of an affection of the spinal cord. This view has received additional support from the results of the post-mortem examinations made by Vulpian and Monakow, to which we have previously referred. Remak mentions, as one of the strongest arguments in favor of the spinal origin of the affection, the fact that the muscles usually in-

---

[1] Mal. du système nerveux, 1879, p. 158. [2] Arch. f. Psych., 1880.
[3] Gaz. méd. de Paris, 19, 1877.

volved belong to a single functional group, while the supinator longus, although it is supplied by the same nerve as the extensors, is not implicated except in rare exceptions. It must be remembered, however, that cases have been reported in which the spinal cord presented no changes.

The theory that the disease is due to an affection of the peripheral nerves is based upon the fact that lesions of the radial nerve have been found, although the spinal cord was intact, and also upon the frequent occurrence of the degeneration-reaction. As we shall see later on, however, this latter phenomenon is not an absolute indication of the peripheral character of the paralysis.

We must therefore conclude that the true pathology of lead-paralysis is still not definitely settled; further investigations may, however, show that the disease is sometimes peripheral, sometimes central in its nature.

The introduction of arsenic into the system also acts as a cause of paralysis, though much more rarely than lead. Like the latter it may give rise to wrist-drop, due to paralysis of the extensors with escape of the supinators. Eulenburg has seen several instances of this kind in workers in artificial flowers. My own experience has been exceedingly limited in this respect, the only case of this kind which has come under my own notice being one of paraplegia following acute arsenical poisoning. As the bladder and rectum were affected in my patient, who is still under observation, I am led to regard the paralysis as probably due to subacute myelitis. The majority of cases of arsenical paralysis assume the paraplegic form. In those cases in which the paralysis is localized, the symptoms with regard to atrophy of the muscles, electrical reactions, etc., are identical with those observed in lead-palsy. As in the latter disease, also, it is doubtful whether the paralysis is due to a lesion of the peripheral nerves or of the spinal cord, and post-mortem examinations are entirely wanting.

Mercurial poisoning is rarely a cause of localized paralysis. The long-continued ingestion or inhalation of mercury gives rise to mercurial tremor, an affection which is not by any means so frequent now as it was in former times. The limbs which are affected with the tremor are always paretic, and in very exceptional cases the tremor disappears and is replaced by localized paralysis of the muscles. Nothing further is known with regard to the electrical reactions and other phenomena presented in the affected muscles, and the pathology of the affection is equally obscure.

Ischæmic Paralysis.

A rare but interesting form of disease is that known as ischæmic paralysis, which is due to an interference with the proper supply of arterial blood to the affected nerves and muscles, either from embolism or occlusion of the vessel from the pressure of an aneurism. Very few cases of this character have been observed, and I shall therefore republish the two following examples, the first one being reported by Rosenthal,[1] the second by Prof. Mannkopf.

Case III.—"A man, æt. 50 years, stated that on October 31, 1869, he was suddenly seized, while walking, with a violent pain in the left leg, rendering motion impossible, and necessitating the removal of the patient,

[1] Clinical Treatise on Diseases of the Nervous System, p. 422.

on the following day, to the Vienna General Hospital. Upon examination, a solid tumor was found in the region of the left obturator foramen, a little larger than a chestnut, pulsating isochronously with the crural artery, and presenting no bruit on auscultation.

Two days afterward I found the left thigh much colder than the right, the movements of extension scarcely appreciable, and the electro-muscular contractility and sensibility considerably diminished in the extensors of the thigh (upon comparison with the corresponding muscles on the healthy side). Upon November 3d (four days after the beginning of the disease), the electro-muscular contractility to faradism was found to be abolished on the anterior surface of the thigh. Gangrene of the limb then set in, followed by chills, and the patient died on November 24th.

Upon autopsy, a sacculated aneurism, as large as a walnut, was found in the neighborhood of the obturator foramen. It originated from the posterior surface of the left crural artery, pushed the vessel upward, and opened into its lumen by an elliptical opening as large as a coffee-bean. The wall of the artery was thickened around this opening, and, on account of the strong tension existing above the neck of the aneurism, the calibre of the artery was narrowed to such an extent that it only permitted the passage of a small-sized sound. The deep femoral artery and the point of emergence of the popliteal artery were obliterated by solid, adherent thrombi."

CASE IV.—"The patient was suffering from an attack of acute articular rheumatism, during the course of which an acute pain suddenly developed in the left calf and foot, accompanied by a sensation of cold in this region. Paralysis of motion and sensation developed in the affected parts, and well-marked degeneration-reaction was present in the paralyzed muscles.

Upon physical examination, mitral insufficiency was found, and there was absence of pulsation in both femoral arteries.

At the autopsy, fibrinous plugs were found at the bifurcation of the aorta in the right common iliac and in the left posterior tibial artery. The spinal cord appeared to be intact; the left tibial nerve, a short distance below its separation from the left peroneal nerve, presented the changes characteristic of parenchymatous and interstitial neuritis; the muscles were found in a condition of myositis."

When the occlusion of the artery is not complete, or when the collateral circulation is established to a certain extent, the paralysis may be intermittent in character.

This was noticed in one of Charcot's patients, in whom paralysis of the right leg occurred from aneurism of the right primary iliac; the paralysis always disappeared during repose.

Physiological experiments have shown that this form of paralysis is due to loss of irritability, occurring first in the nervous structures and then in the muscles, in consequence of the anæmia of these parts. When the abdominal aorta is compressed in animals, paraplegia and anæsthesia of the hind limbs is produced. It has been found, in such cases, that the irritability of the spinal cord is very rapidly lost; that of the peripheral nerves in from three-quarters of an hour to an hour after the aorta has been compressed, while the irritability of the muscles persists for a much longer period.

Syphilitic Paralysis.

In conclusion, we must make a short reference to the influence of syphilis in the production of peripheral paralysis. In very exceptional cases it is said to occur within a short time after the first development of the syphilitic symptoms, and it is probable that in these cases the paralysis is due to the direct action of the syphilitic virus upon the affected nerves. In the majority of cases, however, syphilitic paralysis occurs in the tertiary stages. It is then due to pressure upon the nerves from adjacent periostitis or exostoses, or to the development of gummata in the nerves themselves. The larger number of these paralyses affect the cerebral nerves, especially those supplying the ocular muscles. Sometimes merely a single twig of one of these nerves is implicated, and syphilitic ptosis (paralysis of the levator palpebræ superioris) is perhaps one of the most frequent forms of this variety of the disease. Zeissl mentions a case in which a syphilitic exostosis of the greater sciatic foramen produced pressure upon the sciatic nerve and gave rise to paralysis.

The clinical history of this form is similar to that due to pressure upon the nerves from other causes, and we may therefore refer to the remarks made upon page 166, *et seq.* Careful examination will, however, usually reveal the existence of other evidences of syphilis, especially in its cerebral forms. We have previously entered so fully into a discussion of these symptoms that it is unnecessary to recur to them at this period. The prognosis of syphilitic peripheral paralysis is usually very good, and the disappearance of the disease under anti-syphilitic measures is an excellent diagnostic sign.

CHAPTER II.

DIAGNOSIS AND PROGNOSIS.

THE first question to determine is whether the loss of muscular function present in any individual case is really paralytic in its nature. This can only be done by carefully inquiring into the clinical history, and thus excluding those cases in which the loss of motion is due to disease of the muscles, bones, joints, or ligaments.

Care should also be taken in discriminating between the immobility of a part from loss of power in the muscles, and that occasioned by the production of pain upon movements of the part. This difficulty is frequently experienced in severe cases of sciatica, in which the patients are often unable to move the limb on account of the excruciating agony produced by the slightest movement of the parts.

In infants it is often very difficult to determine the exact location of any paralysis which may be present. This is due to the fact that they are unable to understand our directions, and also because the adipose layer of the skin is frequently so well developed that it hides the presence of muscular atrophy, even though the latter be very considerable in amount. I have very often been unable to detect any difference in the measurements of the limbs in chubby infants, although one of the members was the site of acute infantile paralysis, and the complete loss of power together with the duration of the disease rendered it positive that marked atrophy of the muscles must have occurred.

If we suspect paralysis of any set of muscles in a child too young to obey our orders, we may often determine its presence by holding a bright object in front of him in such a position that the little patient can only reach it by calling into play the suspected muscles. If this cannot be done by the infant, the position of the object should be changed from time to time, and the movements of the little one in the efforts to reach it carefully watched. Some information may also be obtained by performing various passive movements of the parts, and noting the differences in the tonicity of the muscles or in the active resistance which is made.

In some cases of paralysis in infants a mere inspection of the parts will suffice to make a diagnosis, but in others we must not alone go through all the manipulations mentioned above, but must resort to several examinations before we arrive at a definite conclusion with regard to the exact location of the disease. I have seen not a few gross mistakes made in the diagnosis of paralysis in children, and I cannot too strongly enjoin the exercise of care and discrimination in the examination of such patients in whom we suspect its presence.

After having made a diagnosis of paralysis, its character, whether peripheral or central should be then determined. The first inquiry made should be with regard to the cause of the disease. Thus, there can be no question of the character of the affection, if a knife-wound of the

ulnar nerve has been followed by paralysis of the corresponding muscles. In very many instances a careful examination into the etiology of the paralysis will shed full light upon its character, in others no cause can be determined, while in not a few it may have acted upon the central nervous system as well as upon the periphery.

In the majority of cases there are certain distinctive qualities which enable us to distinguish peripheral from central paralysis. In the latter we very rarely find the loss of power limited to a few muscles, and with the exception of the cerebral nerves, the paralysis, as a rule, does not affect an individual nerve, but rather groups of muscles which belong together functionally. In peripheral paralysis, as a matter of course, the loss of power affects those muscles to which the affected nerve is distributed, though, in exceptional cases, the nerve is only partially affected, and only certain of these muscles are involved. In one case, however, which came under my notice, and which I published in the *Medical Record*, January 26, 1878, a spinal hemiplegia was, in all probability, due to peripheral causes. This case presents so many points of interest that I shall republish it:

Case V.—Wm. T., æt. 22 years; patient perfectly healthy until March 8, 1876, when he suddenly felt pain in the left shoulder, which soon extended into the other shoulder and into the cervical and upper dorsal regions of the spine; this was attended with strongly-marked torticollis on the left side. The next day, March 9th, he was admitted to Roosevelt Hospital. For permission to publish his history during his stay in that institution, I am indebted to the kindness of Dr. Wm. H. Draper, under whose care the patient was for a part of the time.

On March 13th a swelling was noticed on the back of the neck, on the left side, and a diagnosis of deep abscess was made. From March 10th to 20th, the patient's temperature varied irregularly from 100° to 104° F.; pulse rapid, and an eruption resembling typhoid appeared on the back and chest; he had severe pain in the back and in the left extremities. From March 20th to April 1st, the patient had complete paralysis of the bladder. He now began to suffer from a feeling of "a tight band" around the lower part of the chest; this lasted eight to ten weeks. Paralysis of motion now began to show itself in the left arm, and soon spread to the left leg; there was also some anæsthesia on the left side of the body. At the end of March paralysis was almost complete on the left side. On April 2d the patient began to slowly regain power over the paralyzed parts; the anæsthesia gave way to hyperæsthesia. For a week succeeding this date there was intense pain on the left side; the left leg and arm are now slightly contractured; some pain in right arm. During the whole illness the temperature did not range above 105°, and was very irregular.

The patient kept on steadily improving, and was discharged from hospital, July 19th. During his sickness he had fibrillary twitchings in the muscles of the left side; was costive from the beginning of the attack; never had headache or other cerebral symptoms; formication and tingling in the left side at times. The patient first came under my own observation about the end of September, 1876. He then had partial left hemiplegia; the left arm and forearm were each half an inch smaller than the corresponding parts on the right side; the movements of the lower limb are much more affected than those of the upper; patient drags *the limb very markedly*, and must use a cane; he has a feeling of numbness

in the adductors of the thigh and flexors of the leg; sensation otherwise normal; reaction of muscles to electricity is normal for both currents. In the left rhomboid minor muscle is found a small, indurated mass, which is very slightly sensitive to pressure.

The patient was treated by local faradization. About the end of December, 1876, the swelling in the rhomboid muscle became tender; it persisted until the middle of January, and then disappeared for a few weeks, to reappear again in various places in the right and left rhomboidei and levatores anguli scapulæ. On February 28th he drew my attention to a small abscess situated over the third and fourth dorsal vertebræ; this was opened, and discharged a small amount of thick pus. On March 1st a very small piece of bone was discharged through the opening, and the next day the patient entered Bellevue Hospital, where Dr. Jacobi laid open a sinus extending four inches upward along the course of the spinous processes; the sinus was probed, but no dead bone was found. On the posterior superior angle of the left scapula I obtained a feeling of crepitus, and the patient winced when this spot was pressed upon. On July 1st he returned to work, feeling quite well. On November 20th he again reported to me with the sinus completely closed, and the swelling and induration in the muscle entirely gone; there is still a slight feeling of roughness over the posterior superior angle of the scapula. The muscular power on the left side is completely restored.

The case is evidently one of inflammation within the spinal canal; the exact location of the lesion is a matter of considerable obscurity. In my opinion the diagnosis lies between meningitis spinalis simplex and peripachymeningitis spinalis. The latter term implies an inflammation outside of the dura mater of the cord, and any paralysis produced by such a lesion must necessarily be of a peripheral nature. My reasons for adopting the latter view are as follows:

1. All of the symptoms of the disease can be readily explained on the theory that it was due to a suppurative inflammation in the cellular tissue between the dura mater and the vertebræ.

2. Some of the characteristic symptoms of meningitis were absent.

a. There were no cerebral symptoms whatever. A case of severe spinal meningitis, especially when the cervical portion of the meninges is affected, without the supervention of any cerebral manifestations, is an exceedingly rare clinical experience.

b. No tenderness was observed along the spinous processes, and none of the rigidity of the spine so characteristic of spinal meningitis.

c. There was absence of increased reflex excitability.

3. Paralysis came on later than is usual in cases of meningitis simplex.

4. In cases of meningitis, attended with as much paralysis as was present in this case, there is usually a coexistent myelitis, and recovery is therefore not so complete as in this instance.

5. If we examine the clinical history carefully we will find that the local symptoms were chiefly confined to the left side of the body. The case was, in reality, a spinal hemplegia, and differs from all others hitherto described in the fact that motion and sensation were affected on the same side. The power of voluntary motion was very much diminished on the affected side, while the disorders of sensation, consisting of formication and tingling, hyperæsthesia of the skin, intense pains, and then numbness, were also confined almost exclusively to the left side (with the exception of a certain amount of pain in the right arm during a short period).

These facts prove conclusively, to my mind, that the diseased process must have produced pressure upon the roots of the nerves on the whole left side of the spinal canal, and while it would be highly improbable that a meningitic process should be limited to the whole length of only one lateral half of the cord, it is readily conceivable that this should take place in the connective tissue surrounding the dura mater, where the disease is not so likely to spread in all directions as on a serous membrane.

A very interesting question presents itself to our notice when we come to consider the etiology of the affection. Was the inflammatory exudation within the substance of the rhomboid muscle the primary cause of the peripachymeningeal inflammation, or was it secondary to this inflammation?

Although it is impossible to absolutely determine the connection between these two trains of occurrences, I think that the weight of evidence and probability is strongly in favor of the first assumption.

A review of the biography of the subject will throw some light upon the question. After very diligent search I have been able to find records of only seven cases of this disease.[1]

I have found that among the seven cases hitherto reported, five were secondary to inflammation outside of the spinal canal, and two were apparently primary. The *a priori* evidence is, therefore, in favor of the secondary origin of the affection in our case.

Positive evidence, however, is not wanting to point in the same direction. In the first place, my patient states that he noticed the swelling in the neck on the first day of his admission to Roosevelt Hospital, thus showing that the inflammation in the external tissues was at least coincident, in point of time, with the beginning of the spinal affection, and in all probability antedated it. Secondly, the discharge of a piece of bone from the fistula, together with the fact that I obtained crepitus over the superior posterior angle of the scapula, in the immediate neighborhood of which the swelling first appeared, is quite conclusive to my mind that the morbid process was originally a necrosis of a portion of the scapula, and that the pus, instead of bursting outward, passed inward, through the intervertebral foramina, and there set up a suppurative inflammation around the dura mater.

The occurrence of trophic changes, especially the development of marked atrophy of the muscles, is also a very valuable sign of the peripheral nature of an attack of paralysis. The only central diseases in which this symptom occurs are acute spinal paralysis of infants and adults (anterior poliomyelitis), progressive muscular atrophy, and glosso-labio-laryngeal (bulbar) paralysis. But these affections are readily distinguished by characteristic symptoms. Acute spinal paralysis frequently begins with fever and symptoms of cerebral or spinal irritation, the paralysis is usually more widespread in the beginning than it is after the lapse of a certain period, and sensory disturbances are generally absent altogether. In progressive muscular atrophy, the course of the disease is slow, the paralysis keeps pace continuously with the atrophy of the muscles, and the latter

---

[1] Mr. John Simon: "Transactions of the London Pathological Society," 1855; Traube: Berlin Medical Society, 1863 (two cases); Mannkopf: Berlin Medicinische Wochenschrift, 1864; Mueller: Ueber Peripachymeningitis Spinalis, Koenigsberg, 1868; Leyden: Klinik der Rueckenmarkskrankheiten (no autopsy); Leyden: Berlin *Klin. Wochenschrift*, December 17, 1879.

usually pursues a definite course, first affecting certain of the small muscles of the hands, then the shoulders or forearms, etc. In this affection, also, sensory disturbances are entirely wanting. In bulbar paralysis the association of facial, glossal, and laryngeal paralysis is so characteristic that the affection cannot readily be mistaken, although a determination of the exact location and nature of the primary lesion may be extremely difficult.

The presence of the degeneration-reaction is another very important sign of peripheral paralysis. This symptom is also observed in the diseases which we have mentioned above, with the exception of progressive muscular atrophy. Erb has, however, reported a case of the latter disease in which this symptom was present. Leyden, in his remarks upon the case of multiple neuritis to which we have referred on page 170 throws out the suggestion that the degeneration-reaction may only occur in spinal diseases when they are accompanied by descending neuritis, and that Erb's case of progressive muscular atrophy may have been of this kind. However this may be, these diseases of the spinal cord are usually easily recognizable, and we must therefore regard the degeneration-reaction as one of the most valuable symptoms of peripheral paralysis.

CHAPTER III.

TREATMENT.

THE therapeutic measures at our command in peripheral paralysis vary with the etiology of the affection, and we shall therefore devote a few words to the treatment of each special variety.

Acute neuritis, if unchecked, is apt to become chronic, and we should therefore endeavor to cut short the neural inflammation as speedily as possible. One of the main indications is complete rest of the affected part, and when this can be effected in no other way, a splint should be employed. The pain, which is usually intense, may be relieved by hypodermic injections of morphine repeated with sufficient frequency to produce comparative ease. Locally we may employ cold, preferably in the form of an ice-bladder, which is applied continuously (it should not be taken off after a few hours, as the latter plan merely succeeds in increasing the local congestion). Whenever the track of the nerve is swollen and œdematous it is advisable to apply a number of leeches along its course. As soon as the inflammation passes into the chronic stage these measures should be discontinued, as they then become useless.

In chronic neuritis we have to deal with a very obstinate affection. In this also we should endeavor to secure rest to the parts, though not so complete as in acute neuritis, on account of the long duration of the disease. Counter-irritation often proves very serviceable, especially when there is any tendency to an upward spread of the inflammation. I generally make use of pieces of fly-blister about an inch square, one of which is first applied at the uppermost portion of the nerve, which is found to be tender on pressure. As soon as this begins to heal another square of the blister is applied directly below it, and this plan is continued until the whole course of the nerve has been treated in the same manner. This is very useful in relieving tenderness of the nerve, and it has also seemed to me to be very efficacious in checking the progress of neuritis ascendens. When pain forms a prominent symptom I often resort to the actual cautery, applied along the entire length of the affected nerve as far as this is practicable. This measure usually produces considerable improvement, but the relief is only temporary, as a rule, and the pain soon returns with its former severity. Another excellent palliative is the use of the hot douche. Several pitcherfuls of hot water (as hot as the patient can bear) should be slowly poured upon the affected parts from a height of a couple of feet; this may be done twice a day for several weeks or even a few months without interruption. This plan is not alone valuable in checking pain, but is also one of the most useful measures at our command for the relief of the contracture of the muscles and the tenderness and partial ankylosis of the joints which are so apt to develop during the course of long-standing neuritis.

Electricity is also indispensable. The galvanic current should alone

be used; the anode may be applied over the course of the nerve, and the cathode at some indifferent point farther up the limb; the current should be applied continuously, strong enough to produce decided redness of the skin and a smart burning sensation at the situation of the electrodes. The application may be repeated daily or every other day, each sitting lasting four or five minutes. So long as the affected nerve manifests pain and tenderness on pressure the electrical current should only be employed in the manner described. I have seen no good effects under such circumstances from applying electricity directly to the paralyzed muscles. The nutritive disturbances in the muscles are due to the condition of the nerves, and we cannot, therefore, hope for much improvement of the paralysis until the inflammation of the nerves has subsided.

The constant galvanic current is not alone useful in relieving pain and diminishing the severity of the neural inflammation, but also in relieving contracture of the paralyzed muscles. After the neuritis has entirely subsided we may apply the electrical current directly to the paralyzed muscles. In those cases in which the faradic current does not cause contraction of the muscles it is useless to resort to this form of electricity. Galvanism should then be employed, the current being interrupted by means of an "interrupter" in the handle of one electrode; interruption of the current (and therefore contraction of the muscles) may also be obtained by stroking the paralyzed muscles with one electrode, the other being held steadily in one position. The current should be merely strong enough to produce visible muscular contractions, the sittings occurring daily or every other day. When the muscles respond to faradism this current should be employed in preference, the application being made directly over the paralyzed muscles. In severe forms of the disease we must be prepared to exercise great patience, and I have not infrequently employed electricity steadily in these cases for periods varying from six months to a year before any decided improvement was obtained.

Passive motion and massage sometimes prove very useful in keeping up the nutrition of the atrophied muscles, in overcoming contracture, and in relieving ankylosis. These latter symptoms may offer some of the most serious obstacles to recovery after the inflammation of the nerve has passed away, and we should therefore direct all our energies toward their removal.

Very little can be done in chronic neuritis in the way of internal medication. The only internal remedies which I have employed in this affection are iodide of potassium and the fluid extract of ergot, either separately or combined. The doses need not exceed fifteen grains of the former or one drachm of the latter. I am unable to make any positive statements with regard to the efficacy of these drugs, as I have always employed them in combination with some of the measures which have been recommended above. I have often thought, however, that they possess a certain remedial value. No mention has been made of the use of opium in any form to relieve the pain of neuritis, because I am firmly convinced that it should only be employed as a last resort in this disease.

Any considerable experience with cases of this disease will serve to dispel the sanguine expectations which may have been formed with regard to the effect of therapeutic measures, but we should not, on the other hand, adopt the expectant plan of treatment. The employment of

persistent and judicious measures will generally produce considerable relief and often furnish very gratifying results.

When the paralysis is due to compression of the nerves by overlying tumors, exostoses, cicatrices, etc., the pressure may sometimes be relieved by suitable surgical measures. In some cases, as, for instance, when a tumor is directly connected with the tissue of the nerve, it becomes necessary to extirpate a portion of the latter. When the excised portion is not too large the cut ends may be brought into coaptation by means of sutures (which are preferably passed through the sheath of the nerve or the connective tissue immediately adjacent), and a proper position given to the limb. It would appear from the unanimous testimony of surgeons that suture of the nerves will not cause immediate union of the cut ends, though it may hasten regeneration. In cases of this kind considerable room is left for the display of ingenuity on the part of the surgeon, and general rules are of very little value.

In ischæmic paralysis nothing can be directly done to remove the offending cause, since no internal medication will hasten in the least the absorption of a clot—in reality, a foreign body—in a vessel. The most that can be done in such cases is to keep the parts warm by the application of bottles filled with warm water, or thick rolls of cotton-batting, and to pay careful attention to the general condition of the patient in order to maintain the circulation. The only chance of recovery lies in the prospect that the collateral circulation may be re-established with sufficient vigor to nourish the affected nerves and muscles before the structure of the latter has been irreparably impaired.

The treatment of toxic paralysis turns chiefly upon that of lead palsy. The iodide of potassium has been employed in this affection since its introduction by Melsens, but the question of its real utility is still undecided. In conformity with the usual practice, however, I have always administered it in small doses in these cases. If it should interfere with digestion it must be discontinued at once, as the disease is usually accompanied by a certain degree of general cachexia, and nothing should therefore be done which would interfere in the least with the nutrition of the body. It is unnecessary to state that tonic remedies should be employed to suit the individual case. Warm baths often prove of decided advantage in lead paralysis, as they tend to hasten elimination of the poison through the integument. Sulphur baths have also been highly recommended for the same purpose, as it was supposed that the lead would combine with the sulphur present in the bath. But this combination could only occur with such portions as had already been excreted, and we therefore believe that the advantage to be derived from a sulphur bath can be obtained with equal readiness from an ordinary warm bath. Electricity also plays an important therapeutical part in the affection under discussion. As a rule, the reaction of the paralyzed muscles to the faradic current is diminished, and in severe cases is entirely lost. In these cases the interrupted galvanic current is indicated, the applications being made daily for a period of five to ten minutes. We should not, however, forget to mention that quite a number of cases have been reported in which the persistent application of the faradic current led to recovery, although the paralyzed muscles did not respond, at first, to this current. This is explained by the fact that faradism increases the irritability of a nerve, even when it is insufficient to produce muscular contraction ; this fact has been proven by physiological experiments. We not infrequently *observe* in lead paralysis that the voluntary power is entirely restored, al-

though the electrical reactions of the affected muscles are still below the normal.

In lead palsy the extensors are kept continuously on the stretch, and this increased tension is an obstacle to the progress of recovery. Various devices have been resorted to in order to overcome this feature, but we shall refer to these measures in the chapter on paralysis of the radial nerve. When very marked atrophy of the muscles has occurred the progress of recovery is usually extremely slow, and in many of these cases a year may elapse before any considerable amount of improvement has occurred. In some instances, indeed, the prognosis is extremely unfavorable, despite the most patient and judicious measures of treatment, and recovery never occurs. Such cases are, however, exceptional.

Rheumatic paralysis sometimes requires little or no treatment. As we have shown in the course of our remarks on the clinical history of this form of paralysis, the milder varieties may occur spontaneously within a period varying from a few days to two weeks. Of course no treatment whatever is required in these cases. In severe forms, if seen within a few days after the beginning of the paralysis, it is perhaps advisable to apply counter-irritation in the form of a fly-blister, as near the *locus morbi* as possible, or to place several leeches in the same position.

Strychnia has been recommended by many authorities in these cases. The high repute of this remedy is undoubtedly due to the fact that it has been frequently employed in those mild forms which recover spontaneously, and the rapid improvement has then been attributed to the use of the drug. For my own part, I may safely state that I have never seen the slightest good effects from the administration of strychnia in any form of peripheral paralysis. Nor do I well see how it could exert any beneficial action. The effect of strychnia is merely to increase the reflex excitability of the spinal cord, and physiological experiments have shown that the irritability of the peripheral nerves remains unaffected by its use.

The good effects of electricity have also been over-estimated, and for the same reason that holds good with regard to the use of strychnia. In severe forms, however, it is the only agent at our command which promises success. The same rules hold good concerning its application as those which we have laid down with regard to lead palsy. In rheumatic paralysis, we should never despair of recovery so long as the slightest muscular reaction is obtained by either current. In fact, improvement may even occur, although the nerves and muscles have ceased entirely to respond to electricity, as I found in one case in which complete recovery was obtained, although all electrical reaction had disappeared for a period of a couple of months. The galvanic current was steadily employed, however, during this entire time, after which faint reactions to this current became apparent.

The treatment of paralyses following infectious diseases is identical with that of the rheumatic forms.

CHAPTER IV.

PARALYSIS OF THE OCULAR MUSCLES.

General Remarks.

We shall devote very little attention to the consideration of paralysis of the ocular muscles, since this affection falls more naturally under the care of the ophthalmologist, and we must therefore refer for fuller details to the standard works on diseases of the eye.

The slightest amount of paralysis in any of the ocular muscles will give rise to a diminished power of motion of the eyeball in the direction which is dominated by the muscle in question. This loss of power gives rise to a diminution of the absolute and of the relative mobility of the globe. The diminution of the former is much slighter than that of the latter, which may be absent altogether when the paresis of the muscle is not well marked. This is due to the fact that in testing the absolute mobility of the eyeball, the paralyzed muscle acts to a certain extent independently of the corresponding one in the other eye, and as the globe is extremely movable, and very slight force is requisite to turn it in one or the other direction, the affected muscle may be sufficiently innervated by a strong effort of the will to perform the required movement. The diminution of the relative mobility, however, becomes evident even when the muscular paresis is very slight. When the patient is directed to look at an object with both eyes the movements of the globes are always associated, and the innervation of the paralyzed muscle, as well as its corresponding healthy fellow, is equal, and merely sufficient to direct the eye whose muscles are unaffected upon the object.

The paretic muscle receives an insufficient amount of innervation, and is therefore unable to carry the globe as far as its fellow. Thus, if we direct a patient whose right external rectus is paralyzed to follow an object, which we hold in the hand, with both eyes, we will find that when the object is held in the right half of the field of vision, it is not followed so far by the right eye as by the left. Upon carefully watching the eye we will also find that the affected muscle appears to act spasmodically, imparting to the eyeball a vibrating movement. In consequence of the insufficiency of the paralyzed muscle, the visual axes of the eyes do not converge upon the object which is looked at, and double vision (diplopia) is therefore the result. This only occurs in that part of the field of vision which is dominated by the paralyzed muscle, and it is also found that the separation of the two images becomes greater, the more the affected muscle is brought into play. This is but natural, since the continued movement of the normal eye while the affected one remains stationary must result in an increase of the divergence of the visual axes. When the eyes are directed into a portion of the field of vision in which the paralyzed muscle is not called into play the diplopia disappears. There *is, therefore,* a distinct portion of the field of vision in which double

vision is observed in paralysis of any of the ocular muscles, and a knowledge of this fact sometimes enables us to make a diagnosis of paresis of a certain muscle when the loss of power is so slight that it produces no apparent visible effect upon the movements of the globe. The variations in the separation of the two images can be best studied by placing a piece of colored glass before one eye, thus enabling the patient to readily differentiate the images.

Very frequently, also, the head is placed in a peculiar position in order that the patient may more readily dominate that portion of the field of vision in which the diplopia is not present. This position is also very significant in making a diagnosis.

Vertigo is an almost constant concomitant symptom of double vision. It was formerly supposed that this was merely due to the confusion of mind caused by the presence of the two images. J. Hughlings Jackson believes that the vertigo from paralysis of ocular muscles "is due to a wrong estimation of the position of external objects by the one eye whose muscle is paralyzed." Thus, if the external rectus is paralyzed, the patient imagines that objects in the outer part of the field of vision are situated more to the side than they really are, and if he attempts to seize an object in this position, he will miss it, his hand passing to the outside. This is due to the fact that in the attempt to retain binocular vision, the paralyzed muscle is innervated more strongly than usual, but without producing an equivalent movement of the globe. The position of objects, however, is determined by experience from the amount of nerve-force which is expended in converging the eyes upon them, and the patient, who expends a large amount of nerve-force upon the paralyzed muscle, therefore thinks that the object is correspondingly far removed from the median line. The apparent motion of surrounding objects which is due to this cause confuses the mind of the patient and gives rise to vertigo. The correctness of this view is substantiated by the fact that the vertigo occurring in ocular paralysis does not disappear when the healthy eye is closed, but only upon closure of the affected one. After the latter has become accustomed to this condition the vertigo will disappear if the healthy eye be kept continually closed.

In long-standing paralysis of the ocular muscles contracture of the antagonists is very apt to develop on account of their unopposed action. This condition is always followed by an extension of that portion of the field of vision in which the double images are visible; it also delays the recovery from the paralysis by keeping the paralyzed muscle in a continual state of tension.

We shall consider separately the clinical history of paralysis of the nerves distributed to the ocular muscles, but the remarks on etiology and treatment will apply equally to all.

Paralysis of the Third Nerve.

The third nerve (motor oculi communis) is distributed to the superior, inferior, and internal recti, the levator palpebræ superioris, inferior oblique, the ciliary muscle, and the sphincter of the iris. The paralysis may involve these muscles separately or in combination. In the latter event the symptoms are very characteristic. The paralysis of the levator labii superioris gives rise to drooping of the upper lid (ptosis), so that the eye is completely closed, and cannot be opened voluntarily. In some ca

however, slight control of the lid is still manifested, but this appears to be due to forced contraction of the corrugator supercilii, which pulls upon the integument, and thus mechanically draws the lid upward. This is readily seen by looking into a mirror and forcibly corrugating the brow; with each movement of the brow, the upper lid will be found to be slightly raised. In complete paralysis of the muscle the patients frequently raise the lid with the fingers. The paralysis of the ocular muscles proper causes loss of the power of moving the globe in the directions governed by these muscles. In this case the patient is only capable of turning the eye outward (contraction of the external rectus), and downward and outward (contraction of the superior oblique). In fresh cases the antero-posterior axis of the eye is situated in its normal position, but in old-standing cases contracture of the non-paralyzed external rectus and superior oblique occurs, and the axis of the eye is directed outward and somewhat downward. The paralysis of the muscles also causes slight protrusion of the eyeball, on account of the loss of the tonicity of these muscles, which exercises traction upon the globe, and thus keeps it well within the orbit. When ptosis is present, diplopia does not occur, as a matter of course. But if the levator palpebræ is not paralyzed, the diplopia becomes excessively annoying, and is present in all parts of the field of vision except to the outside, and downward and outside. The second image is situated above the first and slightly to the inside, and the distance between the two increases as the object approaches the inner limit of the field of vision. On account of the number of muscles paralyzed the position of all objects except those situated to the outside, and to the outside and downward, is miscalculated, and the patients therefore suffer severely from vertigo, so that frequently they are compelled to walk very slowly.

The pupil is markedly dilated (mydriasis) on account of the paralysis of the sphincter iridis. Complete dilatation does not occur, and the introduction of a solution of atropia into the eye will cause the pupil to dilate still further. The iris does not contract to the stimulus of light, since this act is effected by reflex transmission of an impression upon the optic nerve through the motor oculi communis. Distinct vision is therefore interfered with to a certain extent, because the dispersion of light upon the retina is not prevented.

Paralysis of the ciliary muscle causes loss of the power of accommodation. This muscle derives its nervous supply from the ophthalmic or ciliary ganglion, the motor root of which is furnished by a branch of the third nerve. The patients are unable to read small print, and the visual limit for near objects is further removed from the eye; vision is unaffected as regards distant objects.

As we have mentioned in the chapter on etiology, the ciliary muscle may be the only one affected in paralysis after diphtheria.

When all these symptoms are combined it is impossible to mistake their signification, but a diagnosis sometimes becomes difficult when only a single muscle is paretic. The character of the diplopia varies according to the muscle paralyzed. When the internal rectus is affected the images are placed side by side, the false image being situated to the inside of the true one. When the superior rectus is involved double vision is manifested in the upper half of the field of vision—the false image is above and to the outside of the true one. In paralysis of the inferior rectus *these conditions* are reversed. In paralysis of the inferior oblique muscle *the false image* would be above the true one, and is slightly inclined from

the vertical. It is doubtful, however, whether this muscle is ever paralyzed separately.

These relative positions will vary somewhat when more than one muscle is paralyzed, but an accurate knowledge of the action of the ocular muscles will enable us to determine the presence of very slight paresis from the position of the images. Fortunately, however, we are rarely compelled to make a diagnosis in this manner, as the attempt to move the globe in various directions will usually enable us to detect the muscle at fault by mere inspection.

Paralysis of the Fourth Nerve.

The fourth nerve or patheticus is distributed to only a single muscle, viz., the superior oblique. This muscle serves to rotate the eye from without inward, and from below upward, so that the pupil is directed downward and outward. Unless the paralysis is complete, no loss of power in moving the eyeball will be noticeable, as the external and inferior rectus perform the work of the affected muscle. The position of the double images is therefore important in making a diagnosis. Diplopia only occurs in the lower half of the field of vision; the images are situated one above the other, the false image being below the true one, and situated obliquely so that its lower end deviates to the outside. The head is usually inclined forward in order to bring objects into the upper half of the field of vision, and thus avoid the development of double images as far as possible.

Paralysis of the Sixth Nerve.

Like the fourth nerve, the sixth or motor oculi externus is also distributed to a single muscle, viz., the external rectus. This muscle merely revolves the globe outward, and the effects of its paralysis are very simple. Convergent squint is observed from the unopposed action of the internal rectus, and the eye cannot be turned outward.

When the paralysis is slight, strabismus is not present, and the diminished power of the muscle may only become apparent when an object is brought close to the eye. On account of the paresis of the external rectus, its opponent contracts too strongly, and the eye is therefore revolved inward. Diplopia occurs in the outer half of the field of vision, the false image being situated to the outside of the true one, and the distance between them increasing as the objects approach the outer limits of the field of vision. The patient therefore imagines objects to be situated to the outside of their true position.

Etiology.

In my own experience paralysis of the ocular muscles belongs to the more uncommon varieties of paralysis, but this is probably owing to the fact that the attention of patients is often attracted at first by the diplopia, and an ophthalmologist is therefore consulted. The trochlearis nerve is involved much less frequently than either of the others. In paralysis of the motor oculi communis only one or two branches of the nerve are

usually affected, the entire nerve being less often involved; ptosis is frequently the only symptom observed. The paralysis may also affect more than one of the nerves, and sometimes various nerves of both eyes suffer at the same time or in succession.

The affection is most frequently due to rheumatic causes, such as exposure, etc. Patients are, however, very apt to attribute the paralysis to "catching cold," although no definite exposure can be mentioned.

A considerable contingent of the cases is due to syphilis, which probably gives rise more frequently to paralysis of the ocular muscles than of any other; ptosis often develops from this cause. Syphilis may produce lesions of the motor nerves of the eye either within the orbit or in the cranial cavity. In either of these situations it may cause pressure upon the nerves from the development of periostitis, exostoses or gummatous growths growing from the nerves or from adjacent tissues. Within the cranial cavity it may also cause pressure upon the nerves by producing pachymeningitis or basilar meningitis with subsequent retraction of the tissues. These paralyses, as is evident from the nature of the lesion, occur during the tertiary stage of syphilis.

A certain proportion of ocular paralyses are due to various forms of traumatism, such as fracture of the skull in its anterior portions, hemorrhages into the orbit, direct wounds with a knife or other instrument, blows with the fist, etc. A knowledge of the clinical history is always sufficient to enable us to form a correct appreciation of such cases.

Diphtheria and typhoid fever may also be followed by ocular paralysis, the former often giving rise to uncomplicated paralysis of the ciliary muscle (loss of the power of accommodation).

Knapp has reported a case of paralysis of all the ocular muscles due to coal-gas poisoning; this may also be produced by chronic opium-poisoning.

Various intracranial lesions may give rise to the affection, viz., basilar meningitis, periostitis, and exostoses, tumors growing from the base of the skull, aneurisms of the internal carotids. In some cases tumors of the parenchyma of the anterior portion of the brain may produce peripheral paralysis of the motor nerves of the eye by growing downward and thus causing pressure upon the nerves between their exit from the brain and their entrance into the sphenoidal fissure. Tumors of the crura cerebri and pons are also very apt to produce pressure on these nerves.

Diagnosis and Prognosis.

In complete paralysis of all the branches of one of the nerves the appearances presented are sufficiently characteristic to enable a diagnosis to be made by mere inspection, except in paralysis of the trochlearis. In this case, and especially when the paralysis is very slight, the diagnosis can only be made with certainty after a careful study of the relative positions of the double images, as we have shown in the remarks on the preceding page. In doubtful cases of paresis of the other nerves this method should also be adopted.

The mode of development of the paralysis will usually enable us to determine its causation. It may be difficult, however, to ascertain the character of the lesion when it is intracranial in its origin.

In lesions of the base of the brain other cerebral nerves, such as the

fifth and seventh, are apt to be implicated, optic neuritis often develops, there is persistent headache, and paralysis of the limbs does not occur until a later period. When the paralysis is due to the presence of tumors growing in the crus cerebri, hemiplegia of motion and sensation occurs, the paralysis of the motor oculi communis occurs on the side opposite to that of the body, and at a later period the opposite third nerve also becomes affected.

Temporary paralysis of the ocular muscles may occur in the early stages of locomotor ataxia, and it is very important that its character should be recognized. In such cases careful questioning will usually show that the patient has previously suffered from lancinating pains in the legs, that he finds a little difficulty in walking in the dark, and that there is some numbness in the soles of the feet. The absence of the tendon-reflex, as we have shown previously, is also an important diagnostic sign. In addition, one of the pupils may be contracted almost to a pinpoint; atrophy of the optic nerves is also noticed at times in the very beginning of the disease.

The prognosis varies with the nature of the cause. Those varieties which are due to syphilis are usually curable under appropriate treatment, but relapses are not infrequent. Rheumatic cases of recent date also present a favorable prognosis, but the chances of recovery diminish the longer the paralysis has lasted; this is especially true of those cases in which contracture of the antagonistic muscles has occurred.

The paralyses developing after diphtheria and typhoid fever present an excellent prognosis, and recovery often occurs spontaneously.

When the affection is due to traumatism, hemorrhage, etc., the prognosis depends upon the amount of injury which the nerve has sustained, and varies therefore in each individual case. Those forms which are symptomatic of tumors at the base of the brain are, of course, hopeless.

Treatment.

The main reliance must be placed on the prolonged use of electricity; it is immaterial which form is employed, some writers preferring the faradic, others the galvanic current. One electrode should be placed upon the corresponding mastoid process or upon the temple, and the other (with a small, olive-shaped tip) upon that portion of the closed lid which is nearest to the insertion of the paralyzed muscle. The ocular electrode is also applied, at times, directly upon the sclerotic coat, close to the insertion of the affected muscle. This method is often annoying to the patient, and its advantages do not counterbalance its inconveniences. But with either method of application the current employed should be very mild and never sufficiently intense to give rise to pain. Not infrequently a single application of electricity will suffice to produce a decided and permanent improvement in the power of the muscles. It would seem, in fact, as if the passage of the electrical current through the nerves renders the passage of the stimulus of the will more easy.

In paralysis of the levator palpebræ superioris, the ptosis may be overcome and the passive elongation of the muscle relieved at the same time by the application of a small bit of rubber which is fastened to the upper lid and forehead by means of a couple of pieces of adhesive plaster.

The use of prisms, in order to avoid the development of double images,

is not to be recommended, as the separation of the latter varies with the part of the field of vision in which the object is situated. In the secondary contracture of the antagonist muscles, resort must often be had to surgical measures, viz., section of the contractured muscles.

In those forms which are due to periostitis of the bones of the base of the skull, or to basilar meningitis, some benefit may perhaps be obtained by the long-continued administration of iodide of potassium, and the application of counter-irritation to the nape of the neck. In tumors of the brain, unless of a specific nature, treatment is of no avail.

CHAPTER V.

PARALYSIS OF THE NERVE OF MASTICATION.

(Motor Root of the Fifth.)

Clinical History.

This nerve is distributed to the muscles of mastication (temporal, masseter, internal and external pterygoid, mylohyoid and digastric), and to the tensor veli palati; it also sends filaments to the buccinator, but these are purely sensory in character, the muscle deriving its motor supply from the facial nerve. The symptoms of paralysis of the nerve of mastication are well shown in the following case which came under my observation, although it was complicated by a lesion of the sensory branches of the trigeminus, and the symptoms of the latter are therefore superadded.

Case V.—M. E., æt. 45 years, married; no history of specific disease; a moderate drinker. The patient had remittent fever six years ago, and has had chills from time to time ever since. Three years ago he began to suffer from pain in the right temple and the right side of the face; the pain extended to the crown of the head, and was very intense from the beginning. For a period of six months the pain was continuous, but since then, although the patient has pain every day, it comes on in paroxysms lasting three or four minutes, and then disappears for the rest of the day. About a year ago, numbness began to appear in the painful spots and obtained its greatest intensity a week afterward; this has persisted ever since. There has been at times a slight purulent discharge from the right ear, and the patient feels easier when this is running; he has not tasted anything on the right side of the tongue since the anæsthesia appeared in the face.

Present condition.—There is a depression over the right temple due to atrophy of the temporal muscle. All the superficial facial muscles on this side act apparently as well as on the other. There is almost complete anæsthesia on this side, the anæsthetic zone being bounded anteriorly by the median line, posteriorly by a line drawn from the front of the ear to the vertex, and below by a line from the front of the ear to the chin, this boundary running a little above the lower border of the jaw. The integument on the right side of the nose, right upper lip, and half of the cheek adjoining is hard, stiff, thickened, and shining. The right ala nasi is drawn upward, thus enlarging the nostril, which bleeds readily when pricked ; the patient cannot taste anything on the anterior half of the right side of the tongue.

The right lower teeth cannot be carried forward by the patient to a line with the upper row on the right side. When an attempt is made to perform this movement, the lower jaw projects further forward on the left

side than it does on the right, moving apparently as if turned on a pivot around the right temporo-maxillary articulation. The jaws cannot be brought firmly against one another on the right side, and the food cannot be chewed on this side. There is no paralysis of the palate or uvula. I am sorry to state that the electrical reactions of the paralyzed muscles were not taken, as I only saw the patient once, and he then passed out of my observation.

The symptoms of this case are readily explained by pressure upon the trunk of the trigeminus, including the sensory as well as the motor branches (probably from periostitis of the petrous portion of the temporal bone). Pressure upon the former accounts for the neuralgic pain, the anæsthesia and trophic disturbances, while pressure upon the latter explains the motor disorders.[1]

Paralysis of the motor root of the trigeminus is usually unilateral, as in the above-mentioned case, but it is bilateral in rare instances. The motor symptoms described above follow naturally from the loss of power in the muscles of mastication. The masseter, temporal, and the two pterygoids combine in the act of raising the lower jaw, and their paralysis of course abolishes this movement on the affected side. Depression of the jaw is chiefly effected by the action of the digastric and mylohyoid muscles, but their unilateral paralysis is insufficient to prevent this movement, as the corresponding muscles on the opposite side contract with sufficient vigor to produce depression; in bilateral paralysis, however, this movement is also abolished. Lateral movements are effected by the alternate action of the pterygoids and the anterior fibres of the temporal and masseter; these actions are also abolished in this form of paralysis, and the contraction of the corresponding healthy muscles on the opposite side give rise to the peculiar movements which have been described above. As we have previously mentioned, the nerve also sends filaments to the tensor veli palati, but paralysis of this muscle has not been hitherto observed in this affection.

All the various changes in electrical reactions which we have described as occurring in peripheral paralysis, have been occasionally noticed in the affected muscles in this disease; very little attention has been devoted, however, to these symptoms. Atrophy of the muscles usually occurs, and that of the temporals can be readily seen upon inspection; the prominence in the temporal fossa diminishes, and a well-marked depression may even become visible.

Atrophy of the masseter is also readily recognized by placing one finger in the mouth and another upon the cheek over the position of this muscle, and then making a comparison between the two sides.

When the motor trigeminal root is alone paralyzed, the symptoms are restricted to those which we have just described, but some of the other cranial nerves, especially the sensory branches of the fifth, are generally implicated at the same time. The symptoms are then complicated with those due to the concomitant affections.

---

[1] This case is also interesting from a physiological standpoint, as it tends to show that the chorda tympani, which supplies the anterior half of the tongue with the sense of taste, makes its exit from the brain in the course of the trigeminus, as taste was abolished in the distribution of the chorda tympani, although the lesion was undoubtedly situated in the intra-cranial portion of the trigeminus, and there was no peripheral lesion of the seventh nerve, through which the chorda tympani passes during *a part of its course.*

Etiology.

This disease is exceedingly rare, especially as a peripheral affection, but it is occasionally met with in affections of the pons varolii and medulla oblongata.

The peripheral causes which give rise to it are also usually intra-cranial, and include periostitis and exostoses of the bones at the base of the skull, especially the petrous portion of the temporal, aneurisms of the arteries at the base of the brain, and tumors growing in this region. Paralysis of this nerve from a lesion involving its extra-cranial course must be extremely rare, as I have not met with any reported cases of this character. This circumstance is due to the deep-seated position which the nerve occupies after its exit from the cranial cavity.

Diagnosis and Prognosis.

This affection is not readily mistaken for any other, unless a very careless examination is made. The patient usually complains voluntarily of the disturbance in the process of mastication, and inspection of the parts shows that the corresponding muscles are incapable of performing their functions. The peculiar position of the lower jaw when the patient is directed to move it from side to side or antero-posteriorly, is a pathognomonic sign.

The prognosis as regards recovery from the paralysis is bad in all cases; the prognosis as regards life depends upon the character of the primary lesion, whether it is continually progressive, like a tumor or aneurism, or whether its further progress may cease, like that of an exostosis or periostitis.

Treatment.

Very little can be done in this direction. When we suspect that the disease is due to periostitis of the temporal bone, some benefit may perhaps be derived from counter-irritation over the mastoid process, and the internal administration of iodide of potassium. The employment of electricity has also been advised, that current being employed to which the muscles respond most readily. The current must be applied directly to the muscles, as the nerve is so deeply situated that it is not easily reached.

CHAPTER VI.

FACIAL PARALYSIS.

Clinical History.

Facial paralysis[1] is one of the most interesting, as well as the most frequent of all forms of peripheral paralysis. It is unilateral in the large majority of cases, but in exceptional instances it affects both seventh nerves, and is then sometimes known as diplegia facialis. It may develop suddenly, as when the paralysis immediately follows exposure to a draught, or occurs very gradually, as in some cases which are due to pressure on the nerve from a slowly growing tumor, etc. At the onset of the disease, the patients, being misled by the appearance of the parts, often believe that the face is swollen, and are astonished upon being informed of the true condition. When the paralysis is complete, the appearances presented are very characteristic, and cannot be mistaken for any other condition. The wrinkles in the forehead on the paralyzed side disappear, the eye is widely open and staring, the naso-labial fold is effaced, the ala nasi is in closer approximation to the septum of the nose than on the healthy side, the angle of the mouth droops and is nearer to the median line than normal. The contrast between the paralyzed and healthy sides of the face becomes much more marked when the patient attempts to perform voluntary facial movements. The paralyzed side then remains motionless, like a mask, and the healthy side becomes distorted, as those muscles which are inserted into the angle of the mouth draw it over to the normal side because they are unopposed by their antagonists. The eye remains widely open during sleep as well as in the waking condition. When the patient makes a vigorous effort to close the lids the eyeball is rolled upward and slightly inward until the lower border of the cornea is

[1] After emerging from the lateral tract of the medulla oblongata at the lower border of the pons varolii, the seventh nerve passes into the internal auditory canal, and then through the Fallopian canal. It emerges at the stylo-mastoid foramen, immediately beneath the lobe of the ear, passes downward and then forward to spread over the surface of the face. At the first bend (genu) which the nerve makes in the Fallopian canal is situated a gangliform enlargement known as the ganglion geniculatum. From this enlargement emerges the petrosus superficialis major nerve which goes forward to enter the nasal ganglion, after which it passes downward to supply the levator palati, and perhaps other muscles of the velum palati and uvula. It is also supposed that the fibres of the chorda tympani pass through the petrosus superficialis major to *enter* the ganglion geniculatum, after which they pursue the same course as the other fibres of the seventh. The next motor-branch is a small twig which supplies the stapedius muscle. Then the chorda tympani is given off and joins the lingual branch of the trigeminus; it is the nerve of taste which supplies the anterior third or half of the tongue. At the exit of the nerve from the stylo-mastoid foramen, it gives off the posterior auricular branch, which supplies the muscles of the ear. The nerve then divides into its terminal branches, which supply all the muscles of expression and one muscle of mastication, viz.: the buccinator; it also sends filaments to the stylo-hyoid, digastric and stylo-glossus muscles.

on a level with the upper lid. During this attempt the upper lid also becomes slightly lowered, but the mechanism of this action is not clearly understood; it has been supposed that the levator palpebræ superioris, the unopposed action of which causes the lids to be continually open, becomes relaxed during the effort to close the eye. Epiphora, or overflow of tears, is a constant symptom in this condition. This is due to the fact that on account of the paralysis of the lower segment of the orbicularis palpebrærum, especially that portion known as Horner's muscle, the lower lachrymal point is not kept applied against the eyeball, and the tears are not able, therefore, to escape by the usual channel; overflow then occurs as a natural consequence. On account of the exposed condition of the globe of the eye, and the inability to wash away foreign particles by the act of winking, a certain amount of conjunctival irritation is usually present, unless precautions are adopted to shield the eye. A few cases have been reported in which opacity and ulceration of the cornea, etc., occurred, as in cases of severe disease of the trigeminus, but it is extremely questionable whether these symptoms were the result of facial paralysis. The speech of the patient is indistinct on account of his inability to close the lips properly; the imperfection is therefore chiefly noticeable in the pronunciation of labials. He is also unable to whistle, purse the lips, etc., and, in infants, suckling is interfered with; when an attempt is made to distend the cheeks the air escapes through the unclosed angle of the mouth on the paralyzed side, and the cheek flaps as if it were perfectly limp. One of the muscles of mastication, viz.: the buccinator, is also supplied by the facial, and its paralysis therefore interferes to a certain extent with this function. The flaccid condition of this muscle prevents the cheek from being closely applied against the teeth and alveolar processes, and particles of food, therefore, slip in between the teeth and cheek and must often be removed from this position by the aid of the finger.

Some observers state that the tongue deviates to one side when protruded, but this statement is based on an error of observation. The angle of the mouth on the paralyzed side is nearer to the median line than on the sound side, and the tip of the tongue therefore approaches the former. The absence of deviation of the tongue is one of the differential signs between peripheral and cerebral facial paralysis.

In the majority of cases, the uvula and velum palati are not involved, but exceptionally they are also paralyzed on the affected side. The paralyzed half of the velum hangs lower than on the healthy side, and does not contract so readily during phonation; its reflex excitability is also impaired and may be entirely wanting. When the azygos uvulæ muscle is affected, deviation of the uvula occurs; the tip has been found deflected sometimes toward the paralyzed, sometimes toward the healthy side. But certain sources of error should be excluded before making a diagnosis of paralysis of the uvula and velum palati. In the first place, one pillar of the fauces may normally hang lower than its fellow, and thus simulate paralysis; but, in such a case, its curve is found to be sharply defined, and upon irritating the fauces with any foreign substance, it will draw up as forcibly as the opposite one. The uvula also is not unfrequently deflected in health; in some cases, also, when it is long and pendulous, as after any pharyngeal inflammation, it will topple over toward that side to which the face happens to be turned, and may thus simulate paralysis. I saw this mistake made by a distinguished physician of this city, in a case in which the prognosis depended very considerably upon

the occurrence of this symptom, as indicative of the peripheral nature of the affection. Paralysis of the velum palati is supposed to be due to implication of the nervus petrosus superficialis major, which passes from the ganglion geniculatum of the seventh nerve to the spheno-palatine or Meckel's ganglion, and thence to the levator palati and probably other muscles of the velum palati. But this question in physiology is still not definitely settled.

The special senses may also be affected as the result of this disease. Thus, the sense of smell is very often less acute in the nostril of the paralyzed side, though this is not due to any specific influence of the seventh nerve upon the function of smell; its causes are purely mechanical. In the first place, the cessation of the respiratory movements of the ala nasi and the closer application of the ala to the septum of the nose, prevents the introduction of the proper quantity of air, and therefore of a sufficient number of odoriferous particles. Furthermore, the paralysis of the orbicularis palpebrærum and the consequent epiphora result in an insufficient flow of tears through the nasal duct, and then over the Schneiderian membrane. The latter therefore becomes dry and the terminal filaments of the olfactory nerve are, accordingly, not in the proper condition to receive odoriferous impressions.

The sense of taste is also impaired, at times, and the majority of authors agree in the statement that this symptom is quite rare. My own experience has been different, and I have found, after examining a considerable number of patients, that a certain diminution in the sense of taste is present in quite a large proportion of cases taken indiscriminately. Upon testing with various sapid substances, a diminution in the sense of taste will be noticed on the anterior third of the tongue on the affected side. This examination must be conducted carefully, as the patients are apt to give misleading statements. In more exceptional cases, they state that they have various curious subjective sensations of taste in the above-mentioned portion of the tongue. All the symptoms are due to an implication of the chorda tympani (which is contained in the trunk of the seventh nerve during a part of its course) in the primary lesion.

The sense of hearing sometimes presents peculiar disturbances. We do not now refer to those cases in which the paralysis is secondary to a disease of the ear, and in which the auditory phenomena are merely symptomatic of an organic affection. The phenomena in question consist of a disagreeable sensation within the ear, which is experienced whenever the patient hears sounds of any considerable intensity; auditory hyperæsthesia is also present, and is characterized by an increased perception of very high and very low notes. These symptoms were first noticed by Roux upon himself, while suffering from facial paralysis, and have been since confirmed by other observers. Wolff has applied to this condition the term oxyokoia.

These symptoms are attributed to paralysis of the stapedius muscle, which is supplied by a small twig passing off from the facial nerve during its course through the Fallopian canal; the paralysis of this muscle causes increased tension of the membrana tympani on account of the unopposed action of the tensor tympani.

The secretion of saliva on the affected side is usually diminished, and the mucous membrane of the mouth is therefore drier than on the opposite side. Physiological experiments have rendered it probable that the chorda tympani nerve sends secretory fibres to the submaxillary and sublingual glands, and it is supposed that the irritation of these fibres causes

a diminution of the salivary secretion. There is no doubt, however, that the increased dryness of the mouth on the affected side is partly due to the fact that the mouth cannot be completely closed upon that side, and that the consequent exposure to the air causes an increased evaporation of fluid.

The electrical reactions of the paralyzed muscles vary according to the severity of the paralysis. It was in this affection that the degeneration reaction was first observed by Baierlacher. In the most severe cases the irritability of the nerves rapidly diminishes after a few days, and is soon entirely lost to both the faradic and galvanic currents (in some cases it is slightly increased for the first few days). Within a few days the farado-muscular excitability begins to diminish, grows gradually less, and within two or three weeks has entirely disappeared. The galvano-muscular excitability is undisturbed for the first few days, and then begins to increase so that finally the muscles react to a very mild current. The difference between the contractions of the healthy and paralyzed muscles can be very beautifully shown in this affection. If one electrode is placed at some indifferent spot, such as the nape of the neck, and the other over the median line, upon the forehead or chin, so that the electrode covers muscles on both sides of the face, and a mild current is allowed to pass, it will be found that the healthy muscles remain quiet while the paralyzed ones contract vigorously.

Thus, the affected muscles may react to two cells although it requires ten or twelve to produce any response from the healthy ones. After a variable duration of the degeneration-reaction, lasting from a few weeks to several months, it usually begins to disappear, and as the normal galvanic reactions reappear, the farado-muscular contractility also redevelops, and increases until recovery occurs. The irritability of the nerves now also reappears, first to the galvanic and later to the faradic current. Cases of this character usually last at least six months, and often as long as a year, or even more. It is also not very infrequent to find that voluntary power is completely restored, although the electrical irritability of the nerves and muscles is still below the normal.

As the severity of the paralysis varies, in different cases, we may notice all the various forms of changed electrical reactions which we have previously described. The extent of the deviation of these reactions from the normal is usually a good indication of the probable duration of the disease. Those in which the reactions are normal or are only somewhat diminished in quantity, not in quality, will probably recover within a couple of weeks. In exceptional cases, however, these reactions persist for several months, and the disease may run a tedious course. Finally, Brenner has reported cases in which the irritability of the nerves to both currents is heightened for several weeks. It is also stated that electrization of the healthy muscles will sometimes give rise to contraction of the paralyzed ones; this may, however, be due to diffusion of the current. In cases of long standing, and in all incurable cases, the muscles, and apparently also the skin, undergo a certain amount of atrophy. This is frequently so slight that careful scrutiny is necessary to detect it. The entire paralyzed side of the face appears to be smaller than the unaffected, the bones are more prominent, and the integument appears to be thinner. This is not pathognomonic of a peripheral affection, as I have sometimes observed the same condition in facial hemiplegia, which was undoubtedly due to a cerebral lesion acquired in early life. In cerebral facial paralysis of adults these changes are never observed.

Sensory disturbances are extremely rare in the disease under consideration, and their occurrence has been disputed. I have noticed, however, in a few severe cases of rheumatic facial paralysis that the patients complained of numbness of the cheek, and have found that tactile sensation was not as distinct as on the opposite side, though no change could be detected with the æsthesiometer. These symptoms are undoubtedly due to implication of some of the recurrent fibres of the trigeminus by the primary lesion.

An interesting, though infrequent, feature of facial paralysis is the occurrence of spasms and contractures in the affected muscles. The spasms may be either of a tonic or clonic character, and are usually the precursors of contracture.

The muscular spasms generally affect only a few muscles at a time, and may occur spontaneously or during mental emotion, attempted voluntary action, etc. They are not infrequently accompanied by increased mechanical irritability of the parts. When the spasmodic movements are well marked and general, they simulate facial tic.

Contracture of the paralyzed muscles is generally preceded, as we have stated above, by tonic or clonic spasms. Duchenne [1] states that the rapid development of tonicity in a completely paralyzed muscle which has lost its faradic excitability, is an indication that it will become contractured at a later period. Either a single muscle or the entire group of facial muscles may be affected. When all are involved, the eye appears slightly smaller than its fellow, the ala nasi is drawn upward, the naso-labial fold is deeper and higher than the one on the normal side, the upper lip is drawn upward and a little outward, so that a portion of the teeth is continually exposed, and the lower lip is everted. These changes are not so general, of course, when only a few muscles are contractured. In the larger number of cases the affected muscles are also subject to tonic or clonic spasms. This condition not infrequently leads to mistakes in diagnosis, since, at first sight, the deepening of the naso-labial fold on the affected side may lead to the suspicion that the opposite side is paralyzed. I remember a case in which a well-known neurologist of this city fell into this error and mistook a paralysis of the left lower facial muscles for an affection of the right side. At first sight I was also of the opinion that the affection was on the right half of the face, but upon asking the patient to move the muscles, I found that those on the right side could be contracted with perfect facility, while those on the left, and apparently healthy side, were immovable. Upon questioning the patient as to the history of the disease, it became evident that the left half of the face had been at first completely paralyzed, and that contracture developed at a later period. Trousseau also mentions a case of this kind, in which the paralysis was supposed to be on the healthy side of the face by several medical men who had seen the patient. Careful examination of the motility of both halves of the face will, however, always enable us to make a correct diagnosis with regard to the seat of the disease. The contracture of the muscles is probably due to atrophy of the muscular fibres and to retraction of the newly formed interstitial connective tissue, which thus causes shortening. If this process ceases before it becomes excessive, it may entirely overcome the deformity caused by the paralysis, so that no difference is observed in the two halves of the face unless the movements of the healthy side are very marked.

---

[1] Electrisation localisée.

Double facial paralysis (diplegia facialis) is extremely rare as a peripheral affection, though it is not very uncommon as the result of central diseases. But, as we shall see in the chapter on diagnosis, the latter usually affect only the lower facial muscles. The appearances presented in diplegia facialis are very striking. All expression, except that of the eyes, is lost; the natural folds and wrinkles of the face are entirely effaced. The lower lip droops and the saliva dribbles down the chin. The eyes are widely open and staring, and remain so even during sleep. Though the patient laugh ever so heartily, the features remain perfectly immovable. The disturbances of speech are much more marked than in unilateral paralysis, and the interference with deglutition is also very annoying on account of the passage of food between the cheeks and teeth. The electrical reactions vary in no respect from those observed in like cases of unilateral paralysis. The duration and course of the disease is also entirely similar to those already described in the unilateral variety.

Etiology.

The majority of cases of this disease are due to so-called rheumatic causes. They occur from exposure to a draught and sometimes while merely working or playing in the open air. The paralysis develops immediately after the exposure, or a period of one or two days may elapse; in all cases, it attains its greatest severity within a short period after its inception. A considerable proportion of these cases are attended with loss of taste in the anterior third of the tongue on the affected side; the velum palati and uvula are, however, rarely implicated. It is presumed that in this form a certain degree of neuritis develops in that portion of the facial nerve which passes through the Fallopian canal, and that the severity of the paralysis varies with the amount of plastic exudation into the tissue of the nerve.

Light forms of facial paralysis may disappear in from twelve to twenty-four hours, and it is extremely improbable that any inflammatory exudation could be absorbed in this short space of time. We are therefore led to suppose that a simple congestion of the nerve may give rise to temporary loss of power.

Inflammatory diseases of the middle ear or carious or necrotic processes of the petrous portion of the temporal bone in the neighborhood of the Fallopian canal also act as frequent causes of the disease. In such cases the paralysis usually develops slowly, one twig of the nerve being affected after the other. In some of these cases it is probable that the paralysis is due to a direct spread of the inflammation from the middle ear to the adjacent nerve, giving rise to various grades of neuritis or perineuritis. In the milder forms, it is probable, as in mild rheumatic paralysis, that there is simple congestion of the nerve. The sense of taste is frequently implicated in facial paralysis from ear disease, but the velum palati and uvula are rarely, if ever involved. Disturbances of hearing due to the primary aural disease are, of course, always present; but, in addition, the hyperakusis which was described on page 208, is often noticeable.

Quite a number of cases are due to a lesion of the nerve after its exit from the stylo-mastoid foramen. This category includes direct injury to the nerve by a knife or bullet wound, blow upon the face with the fist, a stone, etc., compression of the nerve by the forceps during delivery, the spread of inflammation from abscesses of the parotid gland, implication

of the nerve in new-growths developing in the gland or in the adjacent tissues, division of the nerve by the knife of the surgeon in opening abscesses, extirpating tumors, etc. A characteristic phenomenon in this variety of the disease is the fact that only a few of the muscles are usually involved, and even when all are affected, the paralysis travels gradually from one muscle to the other. This is due to the anatomical arrangement of the nerve-fibres after their exit from the stylo-mastoid foramen, the nerves spreading out and becoming separated from one another. The paralysis is usually severe, the muscles undergo atrophy, present the degeneration-reaction, and often remain paralyzed irremediably; contracture of the affected muscles is not an infrequent result. The chorda tympani is not involved in this variety, and implication of the velum palati and uvula is also absent. Paralysis from pressure of the forceps during delivery was first described by Osiander and Landouzy. When the infant is quiet, it is very difficult and often impossible to detect any paralysis, as the natural folds of the face are very poorly marked in the young. As soon as the child begins to cry, however, the immobility of the paralyzed side and the consequent deformity become very distinct. The affection is usually mild and recovers spontaneously within a few weeks.

Intracranial diseases may also produce peripheral paralysis of the seventh nerve; we exclude from this variety all those cases which are due to an affection of the central nervous system. This category includes basilar meningitis, the exudation in which produces pressure and atrophy of the nerve, periostitis of the petrous portion of the temporal bone, exostoses and tumors growing from the base of the skull, aneurisms of the vessels at the base of the brain. The nerve is paralyzed, in these cases, in its entire distribution. It appears, however, from the history of reported cases (and I can substantiate this by my own experience in several cases) that the sense of taste is not impaired. This fact seems to indicate that the chorda tympani does not leave the brain with the seventh nerve, but enters the latter in some of the numerous anastomoses which it forms with the trigeminus, glosso-pharyngeal and vagus. It is characteristic of this variety of facial paralysis that it is always attended with paralysis of some of the other cranial nerves, or perhaps with irregularity of the pupils, double vision, amaurosis, headache, and later with paralysis of the limbs. The electrical reactions of the muscles vary with the severity of the disease, and present as many variations as are produced in the rheumatic form. Like the paralysis produced by middle-ear trouble, this variety is also produced slowly, as a rule, the loss of power gradually deepening, and one muscle sometimes becoming affected after the other. The disease is naturally very chronic, its recovery depending upon the curability of the primary disease.

Syphilis generally gives rise to peripheral facial paralysis by producing lesions similar to those of the intracranial diseases mentioned above. Thus, it may produce periostitis, exostoses, gummata, or chronic basilar meningitis. In rare instances the syphilitic lesions are situated along the course of the nerve through the Fallopian canal, or even after its exit from the stylo-mastoid foramen. The paralysis is general (affecting all branches of the nerve) and usually develops slowly. The disease generally disappears promptly under appropriate treatment.

Finally, we must refer to the development of peripheral facial paralysis as a sequela of certain of the infectious diseases (diphtheria, variola, scarlatina). These cases are very rare, and their clinical history differs in no respect from that described on page 178.

Diplegia facialis results most frequently from some affection at the base of the brain which gives rise to pressure on both seventh nerves at their exit from the lower border of the pons (basilar meningitis, tumor). It may also be produced by an injury which has given rise to an extravasation of blood into each middle ear, or by the presence of double otitis media. A few cases have also been reported in which it was of a purely rheumatic origin, the paralyses usually developing at an interval of a couple of days. In still another series of cases the affection may have a different origin on the two sides. Thus, it may be due to middle-ear disease on one side, and be of a rheumatic character on the other, etc.

Diagnosis and Prognosis.

The diagnosis of facial paralysis is extremely simple; in addition to noting the absence of the naso-labial fold and the wrinkles on the forehead, the drooping of the angle of the mouth, it is merely necessary to direct the patient to laugh, when the characteristic deformity will immediately appear. In those cases in which contracture of the muscles has occurred, and the folds of the face have therefore been restored, we must, in addition to making a careful inspection of the parts, obtain a history of the beginning of the affection and of the appearances then presented. The exercise of a moderate amount of care in the examination will always enable us to avoid making a mistake in these cases (vide page 210.)

After having made a diagnosis of paralysis, we must determine whether it is of peripheral or central origin. The history of the case is of great importance in this respect. The fact that the affection developed after exposure to a well-defined peripheral cause (exposure, blow, etc.), its occurrence without any cerebral symptoms or without the presence of paralysis in any other portion of the body, the implication of the orbicularis palpebrærum, occipito-frontalis and corrugator supercilii, diminution in the electrical reactions of the nerves and muscles, and, not infrequently, the presence of the degeneration-reaction, the disturbance of taste in the distribution of the chorda tympani (anterior third of tongue), the paralysis of the velum palati and uvula—all these symptoms are valuable indications of the peripheral nature of the affection. But certain central paralyses of the seventh nerve, due to affections of the pons varolii, are differentiated with great difficulty from the peripheral forms. Tumors, hemorrhages, abscesses, etc., of the pons varolii may be symptomatized by facial paralysis, in which all the facial muscles are affected, as in the ordinary peripheral variety, and in which the electrical reactions correspond to those observed in the latter. Two cases of this kind are reported by Rosenthal.[1] As a rule, however, some of the other cranial nerves are implicated either simultaneously with the facial or after a variable period, and, in addition, the limbs become paralyzed on the side of the body opposite to the paralysis (crossed paralysis).

The peculiar anatomical arrangement of the seventh nerve and its branches enables us, in very many instances, to determine the exact site of the lesion which has given rise to the affection.

When the lesion is situated between the exit of the seventh nerve from the brain and the ganglion geniculatum, the paralysis involves all the facial muscles, the velum palati and uvula are implicated, but the

[1] Clinical Treatise on Diseases of the Nervous System, pp. 126 and 127.

sense of taste is not affected; the disturbances of audition (oxyokoioa) to which we have previously referred are also present. The absence of any disturbance of taste appears to corroborate the theory of Schiff, who states that the chorda tympani enters the seventh nerve in the petrosus superficialis major, and any lesion situated above the genu would therefore not interfere with gustation. If the lesion is situated between the ganglion geniculatum and the point at which the chorda tympani leaves the Fallopian canal, the symptoms will vary slightly from those described above. The velum palati and uvula will be unaffected; there will be an interference with gustation, however, in the anterior third or half of the tongue on the paralyzed side. When the lesion is outside of the Fallopian canal, the only symptoms present are those dependent on paralysis of all the facial muscles; in such cases there is no interference with any of the special senses.

In addition to determining the seat of the lesion, we should also endeavor to ascertain its nature. This is usually done with readiness, and is determined by a knowledge of the history of the case and of the pre-existing diseases from which the patient has suffered. Caution must be exercised, however, in pronouncing the affection of a syphilitic nature, as there is a tendency to consider all nervous diseases as syphilitic which occur in a patient who suffers from this affection. It is therefore well to exclude all other causes before attributing the paralysis to syphilis.

The prognosis depends, of course, upon the nature of the cause. When it is due to some irremediable organic lesion, the paralysis will not disappear, and will usually be followed by atrophy and contracture of the muscles. The electrical reactions of the nerves and muscles are a valuable indication of the probable duration of the disease, as we have shown at length in our general remarks on peripheral paralysis. But even in those cases in which the electro-muscular contractility to both currents has entirely disappeared, we should not abandon all hope of final recovery, as a case of this kind has come under my notice in which persevering treatment led to a favorable termination.

Treatment.

In syphilitic facial paralysis, the use of anti-syphilitic remedies alone is sufficient, in the majority of cases, to cure the disease. In those rare forms which occur in the early secondary stage, mercurials are indicated; in the commoner varieties, due to tertiary lesions, the mixed treatment should be employed. The iodide of potassium, as we have so frequently insisted, should be administered in continually increasing doses until the desired effect is produced.

When the disease is due to otitis media or some other affection of the middle ear, the chief attention should be devoted to the treatment of the latter. Great importance must be attached to the frequent use of mild injections into the ear, the treatment of any pharyngeal catarrh which may be present, etc., etc. For further details on this subject we must refer to the text-books on otology. This variety of paralysis is usually very chronic, and electrical treatment, employed in the manner which we shall describe later on, becomes very important.

Surgical measures become necessary where the affection is due to the presence of abscesses, tumors, etc., in the vicinity of the nerve after its exit from the stylo-mastoid foramen. No general advice can be given with

regard to the treatment of this form, as everything depends upon the peculiarities of the individual case. When resort is had to the knife, however, the incisions should be as small and superficial as possible, in order to obviate division of the nerve, regeneration of which does not readily occur in this situation.

In some forms of rheumatic paralysis no treatment is required, as the affection disappears spontaneously within a period of one or two weeks; the majority of cases, however, demand careful attention. Within the first few days it is advisable to place two or three leeches behind the ear and then apply a blister over the mastoid process. It is doubtful, however, whether any decided benefit is obtained from these measures. The use of strychnia is also recommended, but I have never seen the slightest advantage from its administration, and have long since discontinued this remedy. It is advisable to keep the eye covered with a shade in order to prevent irritation from foreign particles. This simple measure often prevents serious annoyance from this cause.

Practically, our only resource consists in the application of electricity. When the muscles react to faradism, this current should be used in preference, an ordinary electrode being placed over the mastoid process, and the other smaller one (about the size of a five-cent piece), being placed over the various paralyzed muscles. The current should merely be strong enough to produce visible muscular contractions, and three sittings weekly, of four to five minutes' duration, are amply sufficient. In milder cases, or when recovery is almost complete, the nerves also react to faradism; in this event, one electrode is placed in the mastoid fossa, and the other is slowly passed up and down the face in a line passing from a point a little behind the outer angle of the eye to the angle of the jaw. In this manner all the branches of the nerve are subjected to the action of the current. When the nerves are insensible to faradism but react to galvanism, the latter current should be employed in the same manner, the positive electrode being placed in the mastoid fossa and the negative passed over the trunks of the nerves. When the electrode is moved to and fro in this way, it is unnecessary to interrupt the current, as the mere passage of the electrode over the muscles suffices for this purpose. When the muscles alone react to galvanism, the positive pole is retained in the mastoid fossa, the negative being placed over the individual muscles (vide Fig. 1). In this case it is necessary to interrupt the current, and this is readily effected by pressing upon an interrupter placed in the handle of one of the electrodes. Here, again, it is merely requisite to obtain a visible muscular contraction. Even when the electrical irritability of the nerves and muscles is entirely abolished, treatment should be steadily continued unless the nature of the cause of the paralysis precludes the possibility of recovery. It is preferable, in these cases, to employ the uninterrupted constant current through the nerves, and the sittings may be held daily.

Dr. Seguin[1] has described an intra-buccal method of applying electricity to the lower facial muscles. The electrode "consists of an ordinary interrupting handle, armed with a rod-like electrode of moderate length, bent at a right angle near its extremity, and terminating in a ball five millimetres in diameter. The whole of the rod or stem, except the ball, should be completely insulated." This may be used upon all the lower facial muscles, the ball being placed on the buccal mucous mem-

[1] Archives of Medicine, Feb., 1880.

brane at parts corresponding to the motor points externally. Dr. Seguin claims to be thus able "in the stage of recovery of rheumatic facial paralysis, to obtain distinct contractions with faradism when the strongest currents which could be tolerated on the skin of the face did not produce them."

It is held that the slow progress of recovery in many cases of facial paralysis, as in various other peripheral paralyses, is very materially retarded and often entirely stopped on account of the elongation of the muscles, which is caused by the unopposed contraction of the antagonists. Various devices have been suggested in order to overcome this feature. The most effective is one recommended by Dr. Van Bibber of Baltimore, who advises the following plan: a metallic hook is introduced into the angle of the mouth, and is connected with a piece of india rubber which is fastened around the ear. By means of this the desirable amount of traction can be exercised upon the angle of the mouth and the elongation of the muscles thus obviated.

The treatment of facial paralysis following acute infectious diseases is identical with that of the rheumatic forms. Various measures have been recommended in the treatment of contracture of the affected muscles, none of which, however, offer much chance of success; these include galvanization of the contractured muscles, the application of faradism to the antagonistic healthy ones, mechanical elongation of the affected parts by stretching them with the fingers, or keeping a rubber ball in the cheek, and even incision of the muscles. The latter plan should not be adopted under any circumstances, as it will only serve to increase the deformity after reunion of the divided parts has occurred.

FIG. 1.—Motor points of the face, showing the position of the electrodes in electrization of the facial nerve and muscles. The anode is placed in the mastoid fossa, and the cathode upon the part indicated in the figure.

CHAPTER VII.

PARALYSIS OF THE SPINAL ACCESSORY.[1]

Clinical History.

This form of paralysis is extremely rare, but the symptoms produced are very characteristic, and agree in all respects with those observed after extirpation of the nerve in physiological experiments. The external or muscular branch is much more frequently affected than the internal. Paralysis of the former nerve gives rise to partial loss of power in the sterno-cleido-mastoid and trapezius.

When the former muscle is paralyzed, the head is drawn by the unopposed action of the corresponding healthy one in such a manner that the mastoid process on the unaffected side is brought closer to the sternum and the chin is carried horizontally toward the paralyzed side. If contracture of the healthy sterno-mastoid develops (and this may occur after the paralysis has lasted for a long time), the head is permanently fixed in this abnormal position. When atrophy of the muscle occurs, the normal protrusion of its belly is lost, and the diminution in size can also be appreciated by rolling it between the fingers and comparing it with the healthy one. The power of flexing the head and at the same time approximating the chin to the opposite shoulder is lost. Paralysis of the trapezius, when accompanied by atrophy, may also be visible to the naked eye upon comparing the muscle with its fellow, especially when the patient is directed to elevate both shoulders. The scapula is lower upon the paralyzed than upon the healthy side, and its posterior border is drawn farther away from the spine on account of the unopposed action of the serratus magnus. The elevation of the shoulder is diminished though not entirely prevented, as the levator anguli scapulæ and rhomboidei are brought more vigorously into play, and thus partially supply the deficiency of the trapezius. In addition, the power of raising the arm above the horizontal is somewhat impaired, as the trapezius aids the serratus magnus in this action by the contraction of its anterior fibres which elevate the acromial end of the scapula. As in paralysis of the sterno-cleido-mastoid, the antagonist muscle (in this case the serratus magnus) may become contractured, and this will result in drawing the scapula permanently downward and away from the spinal column.

More commonly the external branch of the spinal accessory is alone affected; at times, however, the internal anastomotic branch is also in-

[1] The spinal accessory nerve divides into two branches, the internal or anastomotic, and the external or muscular. The former passes to the pneumogastric, and supplies some of the muscles of the velum palati, the constrictors of the pharynx, and all the muscles of the larynx which are concerned in phonation; this branch also possesses an inhibitory influence upon the heart. The external branch is distributed to the sterno-cleido-mastoid and trapezius, but these muscles also receive a portion of their nerve-supply from the two upper cervical nerves.

volved. On account of the fact that the innervation of these muscles is derived from various sources, it is sometimes found that the muscles are not paralyzed in their entirety, but that only a few bundles are affected.

The electrical reactions have not been inquired into very thoroughly, but in most of the cases in which attention has been paid to this point, they were simply diminished. Erb reports a case, however, in which the degeneration-reaction was present. Lesion of the internal branch of the spinal accessory gives rise to paralysis of the velum palati, the muscles of the pharynx, and those muscles of the larynx which are engaged in phonation. In paralysis of both nerves, the velum palati has been found completely motionless; the voice in consequence has a nasal twang, and fluids are apt to regurgitate through the nose. The loss of power in the constrictors of the pharynx causes difficulty in swallowing, the food "sticks in the throat," and the patient exerts unusual effort in forcing it downward. Unilateral paralysis of the muscles of the larynx causes hoarseness and huskiness of the voice, which is not as loud as usual; upon examination with the laryngoscope, the vocal cord on the affected side is found to be motionless during an attempt at articulation, while that of the opposite side often passes beyond the median line. When both vocal cords are paralyzed, complete aphonia is produced. The incomplete closure of the glottis often allows particles of food to enter the larynx, and thus gives rise to paroxysms of cough.

The spinal accessory nerve also possesses an inhibitory influence upon the action of the heart, but no effect is produced upon the character of the pulse when only one nerve is affected; when both are involved, the pulse may be increased in frequency. The following case, which was reported in detail by Seeligmueller,[1] illustrates most of the symptoms to which we have referred.

Case VI.—Amelia F., æt. 24 years; the patient was healthy until nine years ago, at which time she was compelled to carry very heavy vessels of water upon the back. These often spilled, the water running down the neck and back. Shortly afterward, cough and pain developed on the right side. At the same time the neck became very much swollen on both sides, and the patient experienced considerable difficulty in swallowing; the latter symptom did not disappear after the swelling had subsided, and was therefore unconnected with it. The solid food frequently "stuck in the throat," and fluids were regurgitated through the nose. The patient experienced a certain amount of difficulty in speaking, and the respiration was stertorous. Four years ago the preceding symptoms became complicated with gradually increasing weakness of the left arm. About the same time the patient noticed bilateral atrophy in the region of the neck.

Present condition.—Upon inspection, the facial muscles appear normal and can be well contracted. The uvula is not in the median line, but is deflected considerably toward the right side, so that the right arch of the palate is much narrower and higher than that on the left side; the velum palati and uvula remain motionless when irritated or when an attempt is made at phonation. Upon laryngoscopic examination, the rima glottidis is found to be widely opened, and the vocal cords do not change their position during respiration or when the patient is directed to articulate.

The sterno-cleido-mastoids are markedly atrophied, but unequally in

[1] Arch. f. Psych. Bd. III., p. 433.

different portions of the muscles; the bundles which are inserted into the clavicles are very much atrophied, while those inserted into the sternum are somewhat better developed. The clavicular portion of the trapezius has entirely disappeared on the left side, but is retained on the right; the remaining portions of the trapezius are moderately atrophied on both sides. The temperature is normal, but the pulse is somewhat accelerated (ninety per minute).

The left arm is paralyzed; the deltoid muscle is very markedly atrophied, and the other muscles of the limb are also considerably reduced in size.

This case was probably due to a chronic inflammatory process which began around the medulla, compressing the bulbar origin of the spinal accessory nerves, and then spread downward, involving the spinal roots of origin of both nerves, and then also affecting the origin of the left brachial plexus. In a somewhat similar case which came under my own notice, and which was due to an injury to the back of the neck, only the spinal roots of the accessory nerves were affected. This was shown by the fact that the patient could swallow normally and the muscles of the larynx were also in a normal condition. Both sterno-cleido-mastoids were very markedly atrophied, especially those portions which are inserted into the clavicle; the sternal bundles of fibres were also considerably smaller than usual, and the functions of the muscles were correspondingly lost. The trapezii were also very much atrophied, and the power of elevating the shoulders diminished. On the left side, which was more severely affected, the scapula was lower than on the right side, its posterior border was further removed from the spinal column, and the arm could not be raised to the vertical position.

Etiology.

Paralysis of the external branch of the nerve is not very infrequent, and may be due to a great variety of causes; paralysis of both branches, however, is extremely rare. The latter variety may be due to tumors of the posterior cerebral fossa, exostoses in this region, or exposure to a draught, as in a case reported by Fraenkel.[1] Paralysis of the external branch may be caused by blows upon the neck, wounds of various kinds, exposure, pressure of tumors or enlarged glands, and neuritis. It also occurs not infrequently during the course of progressive muscular atrophy. The latter disease is usually regarded as a central process (degeneration of the anterior horns of the spinal column) but within the last two or three years considerable doubt has been cast upon this view of its pathology, and well-known authorities are accepting the doctrine that certa n forms, at least, must be regarded as peripheral in their nature.

Diagnosis and Prognosis.

When the velum palati, constrictors of the pharynx and laryngeal muscles of phonation are paralyzed, there can be no doubt with regard to the affected nerve, as the spinal accessory alone supplies these parts (with

[1] Berl. Klin. Wschrft. I. 1876.

the exception of the velum palati which is also innervated through other paths). Paralysis of the sterno-cleido-mastoid and trapezius muscles is, however, not infrequently mistaken for other affections. Paralysis of the sterno-mastoid may be mistaken for torticollis or spasmodic contraction of the opposite sterno-mastoid. In the paralytic affection, however, the head can be easily restored to its natural position ; in addition, the atrophy of the muscle is usually distinctly perceptible to sight as well as to touch. After the loss of power has lasted for a long time, contracture of the opposite muscle may occur, and a correct diagnosis can only be made, in such cases, from a knowledge of the previous clinical history. Paralysis of the trapezius is most apt to be mistaken for contracture of the serratus magnus, as this will also interfere with the elevation of the shoulder, and will cause the scapula to be depressed and removed from the spinal column. This affection can be readily excluded by the fact that the scapula can be freely moved in all directions by the hand of the physician. Inspection of the parts will often, also, show atrophy of the muscle when it is compared with the opposite side.

Paralysis of the internal branch of the nerve usually presents a very unfavorable prognosis, as the disease, under such circumstances, is due to intracranial or intraspinal processes, which are generally of an incurable character. Fraenkel's patient, in whom the disease was due to rheumatic influences (exposure to a draught), made an excellent recovery.

In paralysis of the external branch the prognosis varies with the etiology. In the rheumatic forms, which are perhaps the most frequent, the chances of recovery are usually good. If the disease has lasted for a long time, however, and is complicated by contracture of the opposing muscles, treatment generally proves unavailing. Those cases which are due to injury, neuritis, or the pressure of enlarged glands, may also recover under appropriate treatment, though improvement usually occurs slowly. When the paralysis is caused by compression of the nerve by means of a tumor, the disease may be relieved if the tumor is situated in such a position that it can be removed by surgical measures.

Treatment.

In all cases, electricity constitutes the chief and sometimes the only plan of treatment. Either the faradic or galvanic current may be employed according as the muscles respond to one or the other. The deep position of the internal branch of the nerve as well as of the muscles supplied by it (velum palati, pharynx, and larynx) prevents the direct application of the current to this portion of the nerve. The electrodes are therefore best applied upon each side of the neck, underneath the angle of the jaw in order to influence the muscles of the velum or pharynx, and upon each side of the larynx to affect the muscles of the latter. Faradization of the sterno-mastoid and trapezius can be readily performed by placing one electrode over the origin of the muscles and stroking various portions of the bodies of the muscles with the other.

In applying the galvanic current to the sterno-mastoids one electrode should be placed over the anterior border of the sterno-mastoid about an inch below the lobe of the ear, and the other at the lower border; to galvanize the trapezius, one electrode is placed over the entrance of the nerve into the muscle (at its anterior border, half-way between the occiput and clavicle), and the other over that portion in which we desire to

produce contraction. When we have reason to suspect a chronic inflammatory process around the spinal roots of origin or compression of the trunk of the nerve by an enlarged gland, counter-irritation by means of fly-blisters or compound tincture of iodine may prove of service. Treatment is usually of no avail when the paralysis is due to an intracranial or intraspinal affection.

When contracture of the opposite sterno-mastoid, or of the serratus magnus occurs, improvement may perhaps be obtained by the steady use of the continuous galvanic current through the contractured muscles. If no relief is obtained in this manner, the former condition may sometimes be relieved by tenotomy of the muscle, and the subsequent application of an orthopædic apparatus to the head in order to prevent a re-development of the contracture.

CHAPTER VIII.

PARALYSIS OF THE HYPOGLOSSUS.[1]

Clinical History.

Paralysis of the tongue is not an infrequent complication of the most varied central diseases, but it is extremely rare as a peripheral affection. The latter may be due to the pressure of tumors growing from the medulla (in which event the paralysis may be bilateral), to a gunshot wound of the neck, or to injury of the nerve during various operations in this region.

In unilateral paralysis of the tongue, the organ when protruded from the mouth, is deflected toward the paralyzed side on account of the unopposed action of the normal genio-glossus muscle. The lateral movements (toward the affected side) are also seriously interfered with or completely abolished. After the paralysis has lasted for a certain length of time, the affected half of the tongue is found to be smaller than the opposite side, the mucous membrane covering it looks wrinkled, and fibrillary contractions may be visible underneath the surface. When the paralysis is bilateral, the tongue lies in the floor of the mouth and cannot be moved in any direction. In such cases, there is marked disturbance of the functions of mastication and articulation. The former is interfered with because the alimentary bolus cannot be readily moved from one side of the mouth to the other by the contractions of the tongue muscles; in addition, the bolus is not forced properly into the fauces nor can the tongue be contracted in such a manner as to separate the buccal cavity from that of the pharynx; in the act of deglutition, therefore, some of the food will regurgitate into the mouth. The articulation of linguals may be entirely abolished, and indeed the pronunciation of all the letters of the alphabet is usually impaired to a certain extent. These disturbances are naturally greater when the paralysis is bilateral and complete, and in such cases it is almost impossible to understand a single word uttered by the patient.

In pure examples of hypoglossal paralysis, there are no disturbances of taste or sensation in the tongue.

The following illustrative case is taken from Weir Mitchell's work on "Injuries of Nerves."

"Alonzo B. Rogers, a colored lad, aged nineteen, was admitted into the Pennsylvania Hospital, November 24, 1871, suffering from a gunshot wound of the neck. The ball entered the left side of the neck, one and a half inch behind and a little below the angle of the jaw.

---

[1] This nerve is distributed to all of the muscles in the infra-hyoid region, which depress the larynx and the hyoid bone after the passage of the alimentary bolus through the pharynx; to one of the muscles in the supra-hyoid region, the genio-hyoid; to most of the muscles which move the tongue; and to the muscular f the tongue itself.

"The tongue was found paralyzed on the left side as regards motion, but not sensation. When protruded, it turned toward the left or wounded side, and could not be held against the upper lip without the aid of the under. When the tip was pressed against the roof of the mouth, it turned toward the left side. The patient could readily press the tip against any point on the right side of the mouth, but on the left the attempt was attended with difficulty. There was no trouble in swallowing, but the patient thought he could not articulate as distinctly as formerly.

"Sensation was not at all impaired. Several tests were made at different times, but the result was always the same. The right side of the tongue readily responded to the electric current, the wounded side did not, but seemed the more sensitive under the current. The wounded side of the tongue was notably atrophied before the patient was discharged. Several attempts were made to find the bullet, but all were unsuccessful. The wound healed without any difficulty, and the patient left December 12, 1871, the paralysis continuing unaltered."

Diagnosis.

The diagnosis of paralysis of the hypoglossus is readily made from a mere inspection of the parts upon directing the patient to move the tongue in various directions. It is sometimes difficult to determine whether the paralysis is of a peripheral or central nature. The most important points in this respect are the etiology of the affection (whether the lesion is situated in the course of the nerve after its exit from the skull) and the implication or non-implication of other nerves, or of one or more limbs. The hypoglossi are affected in glosso-labio-laryngeal paralysis, but this disease runs such a typical course that it is impossible to make a mistake. The prognosis is usually very poor as regards recovery of the paralysis. When the disease is due to an intracranial process, whether central or peripheral, death generally results on account of the secondary implication of other of the bulbar nerves.

Treatment.

Electrical treatment offers the only chance of success, that current being employed to which the muscles respond most readily. The electrodes may be applied directly to the tongue itself, although this is very inconvenient, or one pole is placed above the cornu of the hyoid bone above which the hypoglossal nerve is found in its passage to the tongue.

CHAPTER IX.

PARALYSIS OF THE SERRATUS MAGNUS.

Clinical History.

The paralysis of this muscle, which is supplied by the posterior thoracic nerve[1] (external respiratory nerve of Bell) is extremely interesting on account of the peculiar deformity to which it gives rise. Quite an extensive journal literature has been published on the subject, but the question as to the real cause of the deformity still remains undecided. The paralysis is of comparatively rare occurrence, and is usually unilateral, the large majority of cases having been observed upon the right side. A good idea of the usual course of the disease may be formed from the following history of a case under my observation :

Case VI.—Annie B., æt. 20 years, a servant by occupation; no history of traumatism, though she has been in the habit of lifting heavy weights; no history of syphilis. About four months ago the patient began to suffer from intense darting pains along the outer side of the right arm; these continued for two weeks and were uniformly worse at night; prior to the appearance of the pains in the arm, she also suffered intensely in the anterior part of the right thigh, but these pains only lasted for a few hours. There was no history of any previous exposure. After the neuralgic pains in the arm had lasted for two weeks, she lost the power of raising the arm into a vertical position. Two months later, her friends noticed that the shoulder-blade projected very markedly on the right side, though the patient, who is rather stupid, had been unaware of this fact.

Upon inspection, the patient's hands being held loosely at her sides, very little deviation from the normal can be observed. Upon carefully comparing both scapulæ, however, the inferior angle on the right side is found to be somewhat nearer to the spinal column than on the left; the posterior border is slightly deflected, the upper portion being farther removed from the spinal column than the lower part; the lower angle of the scapula projects very slightly from the wall of the chest; these changes are so slight that they can only be noticed on close observation. When the patient endeavors to raise the arm, the peculiar "angel-wing" de-

[1] The posterior thoracic arises from the fifth and sixth cervical nerves, passes down in front of the scalenus medius muscle, and then along the lateral aspect of the thorax to supply the serratus magnus. The nerve can be most easily reached a little above the clavicle at the posterior border of the sterno-cleido-mastoid. The serratus magnus "arises by nine fleshy digitations from the outer surface and upper border of the eight upper ribs and from the aponeurosis covering the upper intercostal spaces, and is inserted into the whole length of the inner margin of the posterior border of the scapula."

formity starts out in bold relief, and the arm can only be raised to the horizontal position. At the same time that this is done, the posterior border of the scapula begins to move away from the walls of the chest, and when the arm has been raised to the horizontal, the separation of the scapula is so great that almost the entire fist can be introduced between the anterior surface of the bone and the chest-walls. The faradic reaction of the paralyzed muscle was entirely lost in the beginning, and the galvanic excitability was very markedly diminished. The latter current was steadily employed, and the patient advised to exercise the muscle. Within two months recovery had made considerable progress, and when the patient ceased her attendance at the clinic, the cure was practically complete. She again called, after an interval of a couple of months, to report herself entirely well.

The paralysis is very often preceded by neuralgic pains in the arm or shoulder, and sometimes by anæsthesia or hyperæsthesia in these regions. It develops slowly, as a rule, though such a long interval as was apparently present in the case reported above, between the beginning of the affection (neuralgic pain) and the production of the paralysis, is very unusual. Not infrequently other muscles are also affected, such as the infraspinatus, supraspinatus, trapezius, levator anguli scapulæ. The reason for this combination will become evident when we consider the etiology of the disease.

In long standing cases, atrophy of the muscle can be detected if we direct the patient to raise both arms, and carefully compare the appearances presented at the upper and lateral portions of the thorax. If the panniculus adiposus is not too thick, the digitations of the muscle at their origin from the ribs will be found much smaller on the affected side. The electrical reactions to both currents are simply diminished in the majority of cases, though the degeneration-reaction has also been observed.

There is a difference of opinion with regard to the position of the scapula when the arm is held loosely at the side. Some maintain that no deformity whatever is perceptible under these circumstances, while the majority of observers mention the appearances noted in the case which I have reported above, viz., a closer approximation of the lower angle of the scapula to the spinal column, a change in the direction of the posterior border of the scapula from vertical to upward and outward, and slight separation of the lower angle of the bone from the walls of the thorax. In the few cases which have come under my notice, these appearances have always been presented. According to Berger, these changes in the position of the bone are due to the unopposed action of the trapezius, the levator anguli scapulæ and the rhomboidei muscles. The angel-wing deformity, which is produced when the attempt is made to raise the arm, is very difficult of explanation. The simplest view appears to us to be that of Duchenne, which has recently been revived by Lewinski.[1] According to these authors it is merely the result of the contraction of the deltoid. When the arm has been brought into a horizontal plane, it cannot be raised any further by the unassisted efforts of the deltoid. This muscle will therefore exert traction upon the scapula, which is no longer held against the thorax by the serratus magnus; the mere contraction of those

[1] Arch. f. Path. Anat., 1878, pp. 473–500.

fibres of the deltoid which take their origin from the spine of the scapula (the insertion of which into the humerus now forms the *point d'appui*) will naturally cause the posterior border of the scapula to revolve outward and thus become separated from the chest-walls.

The inability to raise the arm to the vertical position also requires a few words of explanation. The serratus magnus muscle is inserted into the entire posterior border of the scapula; when the muscle contracts, it therefore pulls upon the bone and, as the lower angle of the bone is not held so firmly in position by muscular action as the upper, the contraction of the scapula causes the inferior angle to turn forward and upward, and thus, in some sort, to revolve around the superior posterior angle. This causes the anterior angle of the bone to move upward, and the humerus, which is firmly held against the latter by the contraction of the deltoid, is thus pushed upward into the vertical position. This action of the serratus magnus may be readily imitated in cases in which the muscle is paralyzed. The patient is directed to hold the arm steadily in the horizontal position, and the physician then makes forcible pressure forward upon the prominent inferior angle of the scapula; in proportion as the bone is pushed forward, the arm will be found to rise and will finally reach the vertical position.

Erb mentions a case in which a patient, suffering from this affection, could nevertheless raise the arm to the vertical position by bending the chest backward and then swinging the humerus forcibly forward; its inertia carried it past the horizontal into the vertical position, a luxation of the head of the humerus downward being produced at the same time. An exactly similar case has fallen under my own observation.

Baeumler[1] reports a case in which the arm could be almost completely raised into the vertical position by gradual muscular contraction. He found that the deltoid, supraspinatus and infraspinatus muscles were considerably hypertrophied, and by their contraction fixed the arm firmly at a right angle to the axis of the scapula; the trapezius was then brought into action, and by its contraction tilted up the scapula sufficiently to raise the arm almost into a vertical position.

In exceptional instances the paralysis is bilateral, though such cases are usually of a central, not peripheral, origin. A case of this kind has come under my notice in which the paralysis of both muscles was undoubtedly due to a syphilitic lesion of the posterior thoracic nerves. In the patient in question the paralysis of one muscle was well advanced toward recovery when the other became involved. Under the use of anti-syphilitic remedies, the affection was entirely relieved.

The nerve supplying the serratus muscle was called the external respiratory nerve by Bell, under the erroneous impression that the muscle was actively engaged in ordinary inspiration. This notion is refuted by the case to which we have just referred, in which no disturbance of respiration could be detected, although both muscles were completely paralyzed.

Etiology.

The most frequent causes of the disease are traumatism and overwork. For this reason we find that the right side is almost invariably affected, and that it occurs much more frequently in males than in

[1] Deutsches Archiv f. klin. Med. 1880, p. 305.

CHAPTER X.

PARALYSIS OF THE PHRENIC NERVE.

Clinical History.

Peripheral paralysis of this nerve, which is distributed to the diaphragm, is an extremely rare affection; it may be due either to rheumatic causes or to wounds of the nerve in the neck. The diaphragm sometimes loses its contractile power in severe cases of pleurisy or peritonitis, but this is due to a spread of the inflammation from the serous membranes to the structure of the muscle itself, and cannot therefore be regarded as a true paralysis. Paralysis and atrophy of the diaphragm also occur in the last stages of progressive muscular atrophy, and its development is then ominous of approaching death.

The symptoms of this form of paralysis are very characteristic. When the patient makes an inspiration, the epigastrium sinks in instead of becoming more prominent; this is due to the fact that the capacity of the thorax is increased by the contraction of the intercostal muscles and the diaphragm is therefore forced upward to fill up the space. During the act of expiration, the opposite phenomenon is observed, the epigastrium being elevated. During rest, the patient experiences no difficulty in respiration, but as soon as he attempts to perform any active exercise, the breathing becomes labored and the dyspnœa soon becomes extreme, the auxiliary muscles of respiration being called into play. All reflex acts whose performance requires the contraction of the diaphragm, such as coughing, sneezing, etc., are interfered with or entirely abolished, and in this very fact lies one of the chief dangers of the disease. Thus a patient suffering from ordinary bronchitis, whose diaphragm is paralyzed at the same time, will be unable to expectorate the secretion of the bronchial tubes, and is therefore placed in imminent danger of death from suffocation.

The diagnosis of this affection is very simple, as the sinking of the epigastrium during inspiration, and its protrusion on expiration are pathognomonic of this form of paralysis. The cause of the disease is also readily determined from the clinical history of the case.

Treatment.

The treatment consists almost solely of faradization or galvanization of the phrenic nerve or diaphragm. This is effected by pressing one electrode deep into the neck at the anterior border of the sterno-cleido-mastoid immediately above the sternum, and the other along the insertion of the diaphragm into the costal cartilages of the false ribs. In almost all cases the paralyzed muscle reacts well to both currents, but one case has been reported in which the electrical excitability of the muscle was diminished. If the paralysis is due to some inflammatory lesion in the neck along the course of the nerves, counter-irritation is indicated. It is important in these cases to keep the bowels regular and avoid the development of tympanites, as very slight disturbances of this nature will interfere seriously with the proper performance of respiration.

CHAPTER XI.

PARALYSIS OF THE NERVES OF THE ARM.

Paralysis of the Circumflex.

Clinical History.

This nerve supplies the deltoid and teres minor muscles, and sends sensory filaments to the integument of the shoulder; it is very frequently paralyzed either alone or in combination with other nerves. The deltoid raises the arm from the thorax to the horizontal position; the teres minor assists in rotating the arm outward. The paralysis may develop suddenly or very gradually, according to its causation. When it is due to neuritis, it is frequently accompanied by a feeling of numbness or shooting pains in the shoulder; these sensory disturbances are often combined with anæsthesia of the integument in this region. The paralytic symptoms simply consist of the inability of the patient to raise the arm from the side; the action of the teres minor is so slight, and is so thoroughly compensated by other muscles, that we can disregard it altogether. If the lesion of the nerve is a severe one, atrophy of the muscles develops more or less rapidly. When this occurs, the diminished rotundity of the shoulder is readily perceptible, and in extreme cases the finger may be pressed between the humerus and the glenoid cavity of the scapula. In such cases the arm is somewhat longer than its fellow, because the tonicity of the deltoid, which serves to hold the head of the humerus shugly against the scapula, is lost, and the arm therefore droops more than in the normal condition. The electrical reactions vary considerably ; only in the mildest cases are they normal. In the majority of cases which have come under my observation, the excitability of the muscle was simply diminished to both currents; in rarer instances, well-marked degeneration-reaction is observed. I have noticed with special frequency in this form of paralysis that the electrical excitability of the muscle may still be considerably lowered, even after it reacts with normal promptitude to the stimulus of the will.

Etiology.

Traumatism constitutes probably one of the most frequent causes of deltoid paralysis. The injury may be of various kinds, such as a fall upon the shoulder, a blow with a blunt instrument (in one of my patients the paralysis was caused by a blow with the fist, which struck immediately above the clavicle over the course of the brachial plexus), pressure from a dislocated or fractured humerus, pressure from sleeping on the shoulder, or the injury produced in version by the arm during delivery, an example of which has come under my notice. In some of these cases, inflammation of the circumflex nerve may be produced, and, as I have noticed in

several cases, the neuritis may spread upward to the brachial plexus, and thus involve other nerves in the paralysis. Rheumatic influences, such as exposure, also constitute a not very infrequent cause of deltoid paralysis; it may also be produced by overstretching of the muscle. A few cases have been reported in which the affection followed certain of the infectious diseases, such as scarlet fever, small-pox, etc. Finally, deltoid paralysis (usually bilateral) may usher in an attack of lead-palsy, and may be restricted to these muscles, though it generally spreads after a short interval, to the extensors of the forearm. On account of the interesting character of this modification of lead-palsy, I will give a brief abstract of the history of a patient now under my observation in Randall's Island Hospital :

Case VIII.—Laurence Clooney, æt. 37 years, house painter; entered the hospital, May 13, 1880. He has worked at his trade for eighteen years; two years ago he had an attack of lead colic. The patient continued well since then until January 6, 1880, when he suddenly noticed while at work that he could not lift his arms from the side (paralysis of the deltoids). Two days afterward he again had an attack of colic, and pains began to be felt in both arms. He kept on working until April 25th, when, on attempting to wash his hands, he found that they were also paralyzed; two days later an attack of colic occurred, and five days later another attack.

Present condition.—The patient has a sallow-complexion ; he thinks he has lost flesh ; marked blue line on the gums. No loss of sensibility can be discovered in any part of the body. The deltoid muscles are completely paralyzed (he is unable to lift the arm from the side of the chest) and are markedly atrophied, the right to a greater extent than the left; he presents weakness with some atrophy of the biceps ; the triceps is not appreciably affected. Paralysis of the extensors of the wrist, more marked on the right side, with very considerable atrophy of these muscles is also noticeable. The supinator longus of the right arm is paretic and smaller in size than the left. All the affected muscles present a diminished reaction to the faradic current.

Diagnosis.

The diagnosis of deltoid paralysis is very readily made from the loss of function of the muscle. In two of my cases, occurring in a child (in whom all forms of paralysis are detected with much greater difficulty than in the adult), a diagnosis had been made of dislocation of the humerus. This mistake is readily obviated by placing the hand of the affected side over the opposite shoulder. In paralysis of the deltoid, the elbow can be brought in apposition with the chest, while the arm is in this position ; in dislocation of the humerus this cannot be done. Another patient was referred to me by a surgeon, who thought the case was one of deltoid paralysis. A very slight examination, however, served to show that the patient was suffering from fracture of the humerus. A mistake of this kind can only result from sheer carelessness.

In rare instances infantile paralysis affects the deltoid without involving any other muscles, and it may then be very difficult to decide whether the paralysis is of a peripheral or central nature. The distinguishing fea-

tures of the latter are : its occurrence in childhood, the frequent development of febrile symptoms prior to the paralysis, the rapid atrophy of the muscle, the absence of any determinable cause, and the almost absolutely unfavorable prognosis as regards recovery from the paralysis.

Treatment.

Electricity furnishes our chief resource in the treatment of paralysis of this nerve. As a general thing that current should be employed to which the muscle responds most readily, but it is found, in exceptional cases, that recovery occurs under the continued employment of faradism although the paralyzed muscles do not react to this current. In applying electricity to the deltoid, one electrode should be placed upon its anterior fibres below the clavicle and the other slowly moved across the body of the muscle. This method is applicable to both currents, and does not require the use of the interrupting handle. When the paralysis is due to neuritis, which is still present in an active stage, it is doubtful whether electrization of the muscle itself is attended with much benefit. In such cases it is well to pass the uninterrupted constant current through the nerve, one electrode being placed upon the brachial plexus in the neck, and the other near the insertion of the muscle into the humerus. There appears to be a special tendency for neuritis of the circumflex to spread upward and secondarily involve other branches of the brachial plexus. Counter-irritation (in the form of fly-blisters) is useful in combating this tendency; the blisters should be applied upon any painful spots which may be present in the muscle, and also along the course of the plexus in the root of the neck (at the anterior border of the trapezius).

When the paralysis is due to pressure upon the nerve from dislocation or fracture of the humerus, etc., the appropriate surgical measures must, of course, be adopted.

Paralysis of the Musculo-Cutaneous Nerve.

Clinical History.

This nerve supplies the biceps, brachialis anticus, and coraco-brachialis muscles, and is also distributed to the integument of the outer and anterior aspects of the forearm and ball of the thumb, and the lower third of the posterior surface of the forearm. It is very rarely paralyzed after leaving the brachial plexus, as it is situated very deeply and therefore not liable to injury. Even when a trauma affects the plexus, this nerve usually escapes on account of its protected position. I have a case under observation, however, at present, in which this nerve alone became paralyzed while the individual was on a drunken debauch ; the patient is unable to give an account of the nature of the accident, but it probably occurred from injury of some kind.

The symptoms of this form of paralysis consist merely of partial loss of the power of flexing the forearm upon the arm and loss of sensation on the anterior and posterior aspects of the outer side of the forearm. Flexion of the forearm is not entirely lost because the supinator longus

acts as flexor as well as supinator. As this disease usually forms part of paralysis of the entire brachial plexus, its etiology and treatment are similar to those of the latter. (Fig. 3.)

Paralysis of the Median Nerve.

Clinical History.

This nerve supplies the deep and superficial flexors, the flexor carpi radialis, the muscles of the ball of the thumb with the exception of the adductor pollicis, both pronators, and the first three lumbricales; it is also distributed to the integument of the anterior surface of the thumb, the first two fingers and outer half of the ring finger, and the dorsal aspect of the second and third phalanges of the index and middle fingers and outer half of the ring finger.

Paralysis of the deep and superficial flexors causes loss of the power of flexion of the second and third phalanges; it does not affect the first

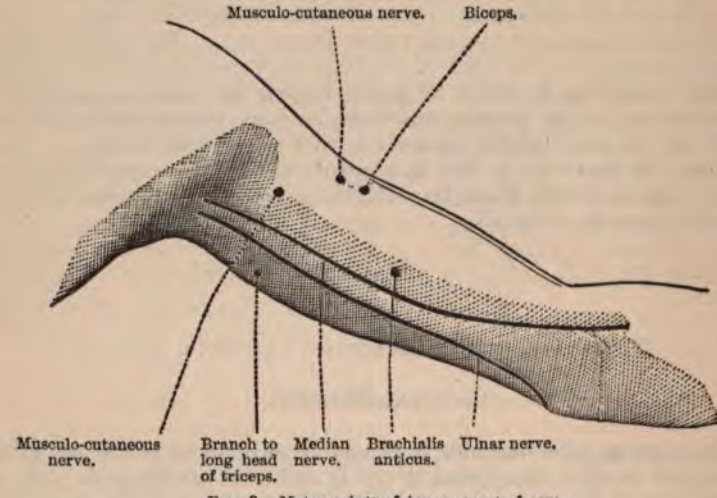


Fig. 3.—Motor points of inner aspect of arm.

phalanges as they are flexed by the contraction of the interossei muscles, which, at the same time, produce extension of the second and third phalanges. When the paralysis of the flexor muscles is complete and long-continued, the unopposed action of the interossei may permanently cause flexion of the first phalanges and hyperextension of the second and third. Paralysis of the pronators of the forearm (which are also supplied by the median nerve) interfere with pronation to a certain extent, though not completely, as this function is performed in part by the supinator longus, when the forearm is flexed upon the arm.

Flexion of the wrist cannot be performed normally, on account of the paralysis of the flexor carpi radialis. The paralysis of the muscles of

the thumb, with the exception of the adductor pollicis, causes loss of the power of touching the tips of the fingers with the thumb and of pressing the thumb firmly against the forefinger. The unopposed action of the extensor longus pollicis produces hyperextension of the thumb, and at the same time cause a disappearance of the natural projection of the ball of the thumb anteriorly, thus flattening the entire anterior surface of the hand.

Where the entire trunk of the nerve is wounded, sensation may be lost on the anterior surface of the thumb, the first two fingers, outer half of the ring finger, the dorsal aspect of the second and third phalanges of the index and middle fingers, and the outer half of the ring finger. The distribution of the sensory disturbances, is however, subject to very great modification, and indeed Richet has reported a case (to which we have previously referred) in which division of the median nerve was not followed by any loss of sensation in the hand. This peculiar phenomenon is explained by the presence of Arloing and Tripier's recurrent sensory fibres, which pass from the ulnar and radial nerves to the median, and to which reference has been so frequently made in the article on neuralgia. Various trophic disturbances, which are entirely similar in all respects to those which have been mentioned as occurring in traumatic neuralgia of the upper limbs, are also observed in severe paralysis of this nerve.

Etiology.

Median paralysis is due to a great variety of causes, especially to traumatism, including gunshot wounds, incised wounds with a knife, pieces of glass, etc., blows with a club; to pressure from tumors of various kinds, from excessive development of callus after fracture, from the bands of a strait-jacket when drawn too tightly; from neuritis in consequence of exposure, or as a sequel of certain infectious diseases.

This nerve is, however, rarely paralyzed separately and, indeed, it is unfrequently met with even in combination with paralysis of other nerves of the arm.

Treatment.

The remarks made on the treatment of deltoid paralysis also hold good with reference to this form of paralysis. In applying electricity to the median nerve, one electrode is placed in the fold of the elbow to the inside of the median line (vide Fig. 4) and the other electrode is applied above the wrist directly in the median line. The muscles of the ball of the thumb and the lumbricales may be electrized through the nerve by placing one electrode over the lower portion of the nerve above the wrist, and the other (a small olive-pointed electrode) upon the motor points shown in Fig. 4, or the current may be passed through the muscles (usually more effectual), the olive-pointed electrode being retained in the position just described, and the other being placed on the dorsum of the hand.

Surgical measures frequently prove of decided relief when the paralysis is due to compression of the nerve by an old cicatrix, a foreign body in a wound, etc. These cases are often attended with considerable pain, which is sometimes so severe as to necessitate the hypodermic administration of morphine.

Paralysis of the Ulnar Nerve.

Clinical History.

The motor fibres of the ulnar nerve are distributed to the flexor carpi ulnaris and a portion of the flexor digitorum profundus, the interossei (both palmar and dorsal), first two lumbricales, the adductor pollicis and the muscles of the hypothenar eminence; the sensory fibres are distributed to the ulnar third of the palm of the hand, the flexor aspect of the

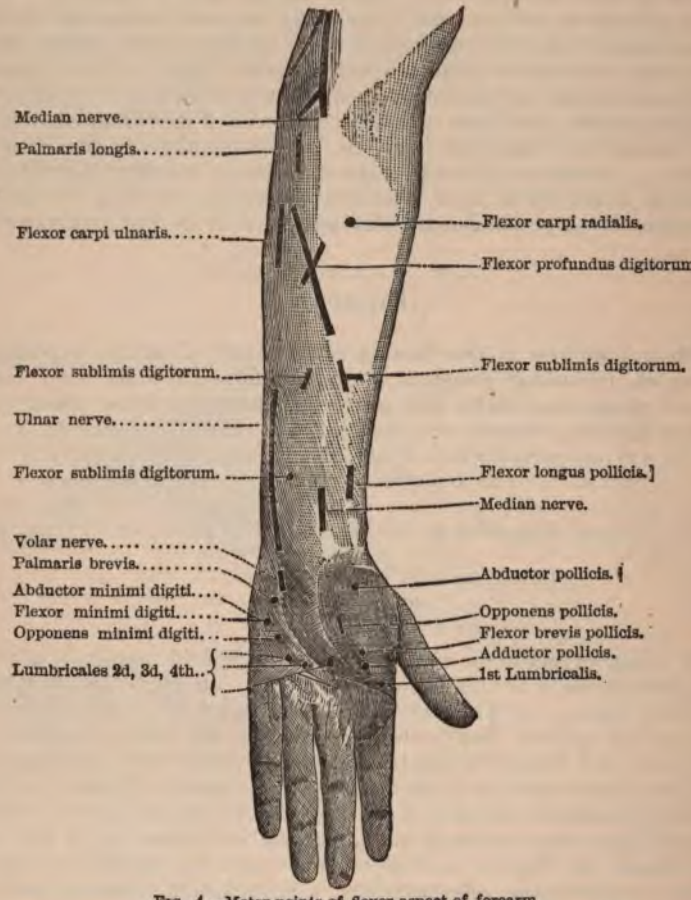


Fig. 4.—Motor points of flexor aspect of forearm.

little finger, ulnar half of the ring finger, and the dorsal aspect of the little and ring fingers and the ulnar half of the middle finger, with the exception of the first phalanx, which is supplied by the median nerve.

Paralysis of this nerve, therefore, leads to partial loss of the power of flexion of the wrist and fingers. The paralysis of the interossei causes loss of power of adduction and abduction of the fingers, and also of flex-

ion of the first phalanx and extension of the second and third phalanges. When the paralysis is complete and long-continued, a very peculiar and characteristic deformity is produced, on account of the unopposed action of the antagonists of the interossei, viz., the superficial and deep flexors, which flex the second and third phalanges, and the common extensor, which extends the first phalanx. The contraction of the latter muscle causes hyperextension of the first phalanges; the action of the former produces complete flexion of the second and third phalanges, thus giving rise to what is known as the "claw-hand."

In addition, paralysis of the adductor pollicis causes diminution in the power with which the thumb can be pressed against the index finger; paralysis of the muscles of the hypothenar eminence interferes with the various movements of the little finger. In severe forms of paralysis, the muscles undergo atrophy, and this is very readily detected when the dorsal interossei are affected; the metacarpal bones then project prominently and depressions are visible between them.

There may be considerable loss of muscular power without any affection of sensation; when present, the latter usually affects the integument in the manner referred to in describing the distribution of the nerve, but variations are also observed in the case of this nerve, though not so marked as in paralysis of the median. The only variation which has come under my own notice has been that the loss of sensation has extended over the outer half of the flexor aspect of the ring finger, and has affected half instead of a third of the palm.

When the paralysis is due to an irritative lesion of the nerve, it is not infrequently complicated with numbness and tingling, or lancinating pains in the anæsthetic regions. The trophic and vaso-motor disturbances which are noticeable are similar to those occurring in other paralyses of the arm.

Etiology.

This form of paralysis is due in the majority of cases to traumatic influences, to which the nerve is often subjected on account of its exposed position. These include blows upon the forearm, sleeping on the arm, wounds with a knife or other sharp instrument, pressure of a straight-jacket, pressure of a crutch upon the nerve in the axilla. It is also produced by neuritis due to exposure or to the propagation of inflammation from surrounding parts, and to the pressure of a neuroma or other form of tumor.

The peculiar claw-shaped hand is not pathognomonic of this form of paralysis, but is also observed at times in progressive muscular atrophy. But other characteristic symptoms then serve to differentiate the two affections. Thus, in the latter the atrophy often begins in the muscles of the ball of the thumb, it gradually spreads up the arm, and then travels to the trunk and lower limbs, involving the muscles irrespective of their nervous supply, sensory disturbances are entirely absent and the loss of power keeps pace *pari passu* with the muscular atrophy.

Treatment.

The treatment of ulnar paralysis is similar to that of the other nerves of the arm. Electricity is employed in this affection by applying one electrode upon the fold of the elbow over the inner condyle, and the

other upon the inner side of the wrist (Fig. 3). The interossei are electrized by placing one electrode over the nerve at the wrist and the other over the motor points of the muscles as shown in Fig. 4, or by placing one electrode in the palm in the manner described on page 235, in speaking of electrization of the lumbricales.

When there is a tendency to the development of the "claw-hand," the contracture of the muscles should be overcome by the application of a straight splint to the palm of the hand and fingers. Galvanization of the opposing muscles is also said to be useful under these circumstances. Too much must not, however, be expected from the employment of these measures, as the peculiar deformity may develop despite all treatment, and render the hand entirely useless.

Paralysis of the Musculo-spiral Nerve.

Clinical History.

During its course through the spiral groove of the humerus, this nerve is distributed to the triceps, anconeus, supinator longus, and extensor carpi radialis longior; it also supplies the integument of the outer and posterior aspect of the lower half of the arm, and the upper two-thirds of the posterior aspect of the forearm. The nerve divides into the radial and posterior interosseous nerves, the former being distributed to the integument of the outer half of the dorsum of the hand and the dorsal aspect of the thumb, the index finger and outer half of the middle finger, with the exception of the third phalanges (which are supplied by the median). The posterior interosseous nerve is distributed to all the muscles on the posterior aspect of the forearm.

This form of paralysis of the nerves of the arm is much more common than the varieties previously described, and also differs from the other varieties in the fact that it very often occurs separately, though the branch above the elbow is rarely involved except in combination with other nerves of the brachial plexus.

The branches supplying the triceps and the integument of the arm are so rarely involved apart from paralysis of the other nerves of the plexus, that it is unnecessary to consider them in this connection.

Paralysis of the extensors gives rise to the characteristic appearance known as wrist-drop, the hand and first phalanges being flexed, the second and third phalanges extended. The patients are unable to extend the wrist voluntarily, and for this reason the flexors of the forearm are also apparently weakened (vide page 182). When the paralysis is complete and affects both arms, the patients are rendered almost entirely helpless, as the loss of power of the extensors not only prevents the performance of extension, but the inability to steady the wrist also interferes with flexion of the fingers. Paralysis of the supinator longus causes partial loss of the power of supination of the forearm (this is partially effected by the biceps) and also, to a certain extent of flexion, as this function is assumed by the supinator when the forearm is maintained in a semipronated position. A very characteristic appearance is presented when the paralysis is attended with muscular atrophy; the prominence upon the outer side of the forearm, produced by the bellies of the extensor muscles is lost, and in extreme cases the finger may be pressed deep into the posterior interosseous space.

In lead paralysis, as we have previously pointed out, the supinator longus is only exceptionally affected, and we therefore find that this muscle stands out prominently, while the other extensors may be wasted to such an extent that they are detected with difficulty.

This form of paralysis has appeared to me to be more frequently complicated with trophic disturbances than the other paralyses of the nerves of the arm.

Its etiology embraces the entire list of causes which have been described as productive of ulnar and median paralysis. In addition, lead palsy is usually localized in the distribution of this nerve. The majority of cases of musculo-spiral paralysis, however, are due to sleeping upon the arm, allowing it to hang over the edge of a chair, etc. Among upward of twenty-five of my cases due to the latter cause, in all but two the paralysis developed while the patients were sleeping off the effects of a heavy debauch. It occurs with so much greater frequency under these conditions, because the patients sleep more heavily and change their position less frequently. Gowers [1] reports two cases in which paralysis of the musculo-spiral nerve occurred from violent contraction of the triceps muscle.

Diagnosis.

The diagnosis of this form of paralysis is readily made from the position of the hand and the inability to perform extension. It is often difficult, however, to determine whether the paralysis is due to lead poisoning or to other causes. In the former event, there is usually a history of the entrance of lead into the system in some manner, the patient has suffered from one or more attacks of lead colic, a blue line is present upon the gums, and the supinator longus is unaffected in the large majority of cases. In addition, the paralysis generally involves both arms, and, in fact, any bilateral paralysis of the extensors, in which a cause cannot be determined, should lead us to suspect the presence of lead in the system,

In one case under my observation bilateral paralysis of the extensors was due to rheumatic influences, viz.: exposure to a high wind. The patient, a washerwoman, hung clothes upon the roof in a strong wind, her arms being wet at the time, and upon the following morning awoke with wrist-drop in both arms. The disease was differentiated from lead-palsy by the fact that there was no history of lead-poisoning, no blue line on the gums, the supinators were affected to the same extent as the extensors, and the paralysis ensued shortly after the action of a sufficient exciting cause.

In rare cases some difficulty is experienced in differentiating the disease from progressive muscular atrophy, but the latter is generally accompanied by atrophy of muscles in other parts of the upper limbs, especially in the interossei and muscles of the ball of the thumb.

Treatment.

The treatment is, in general terms, the same as that of ulnar and median paralysis. The extensor muscles are electrized by placing one electrode over their general origin from the external condyle of the humerus

[1] Medical Times and Gazette, 1877, p. 475.

and gently stroking the posterior surface of the forearm with the other; in this manner all the muscles are successively brought into play, and this method may be employed for both currents. (Figs. 5 and 6.) Galvanization of the nerve may be effected by placing one electrode in the fold of the elbow over the external condyle and the other at the back of the wrist in the median line. In the treatment of lead-palsy recovery is sometimes obtained under the use of the faradic current, although the muscles no longer respond to this form of electricity. In all cases of lead-paralysis, however, it is more advisable to use the interrupted galvanic current.

When wrist-drop develops, the extensor muscles, as we have previously seen, are put upon the stretch, and this condition interferes, to a certain extent, with the progress of recovery. Various devices have been resorted to in order to obviate this difficulty, the simplest being that invented by Dr. Van Bibber, and which may be modified in various ways.

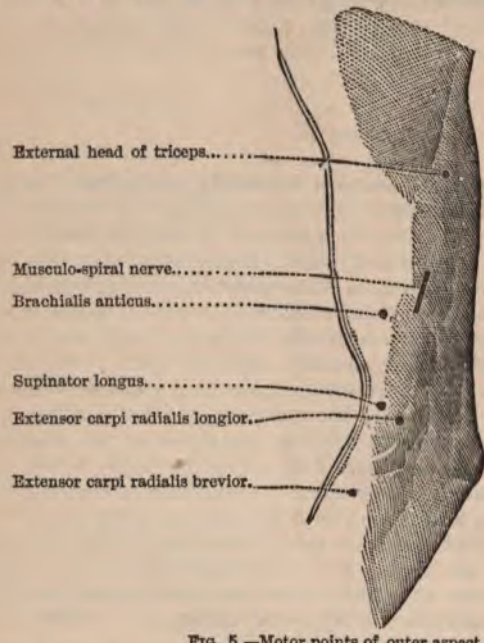


FIG. 5.—Motor points of outer aspect of arm.

A fingerless glove is worn upon the paralyzed hand, into the back of which is inserted a wire loop in the median line. Another loop is inserted over the posterior surface of the elbow by means of a piece of moleskin plaster, which adheres to the back of the arm; an artificial muscle, consisting of simple rubber tubing, is then introduced between the two wire loops, and should be drawn sufficiently tight to keep the hand continually in slight extension. A rubber ring or band of any description may be placed around the wrist (allowing the artificial muscle to pass between it and the skin), thus preventing the apparatus from rubbing against the clothes and becoming displaced. When the apparatus has been properly adjusted the patient is often enabled to perform movements with the hand which were previously impossible, and the relief of the muscular tension undoubtedly facilitates recovery.

Whenever this form of paralysis (as well as all the other varieties of paralysis of the nerves of the arm) is attended with trophic changes in the joints (pain, swelling of the ends of the bones, ankylosis), considerable benefit is often derived from the persistent use of hot douches, applied half an hour daily. In severe cases the constant galvanic current may be

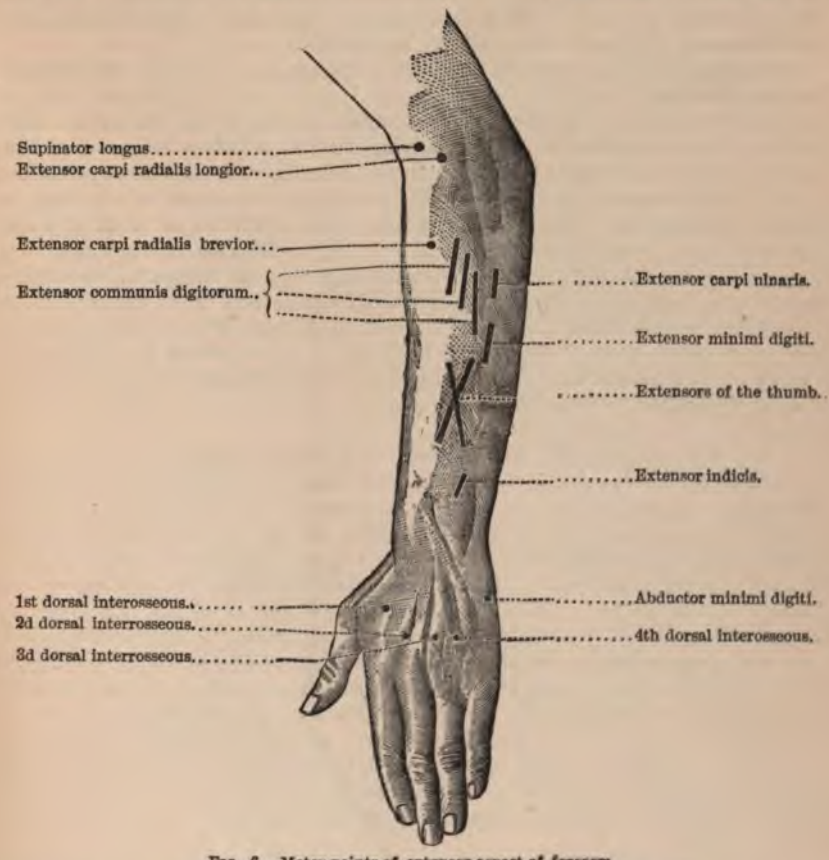


FIG. 6.—Motor points of extensor aspect of forearm.

employed, the current being passed directly through the affected joint, and a sufficient number of cells being used to cause considerable pain and decided redness of the integument. The applications should be made daily, the duration varying from ten to fifteen minutes.

In lead-paralysis constitutional treatment is also required, but this has been referred to with sufficient detail on page 194.

COMBINED PARALYSIS OF THE NERVES OF THE ARM.

With the exception of paralysis of the circumflex and musculo-spiral nerves, which often occur separately, several of the nerves of the brachial plexus are usually affected at the same time. This is due to the fact that

the nerves are situated so closely together in their passage down the neck and arm, and also to the tendency—more marked in this than in any other part of the body—of the spread of neuritis along the course of the nerve-trunks, thus leading to the secondary implication of other branches of the plexus. As we have shown in the course of our general remarks on peripheral paralysis, neuritis descendens as well as ascendens are observed in the nerves of the upper limbs, and in some cases, indeed, the inflammatory process extends to the spinal cord.

Erb first described a peculiar form of paralysis in which the deltoid, biceps, brachialis anticus and supinator longus were paralyzed, and, at times, the muscles supplied by the median nerve in the forearm. Erb believed that this form of paralysis was due to a lesion situated at the exit of the sixth cervical nerve from the scalenus muscle, as the application of the faradic current to this spot will produce contraction of the above-mentioned muscles. These observations have been since confirmed by Ernst Remak [1] and H. ten Cate Hoedemaker.[2] As Erb has pointed out, the same group of muscles are sometimes affected in the paralysis of the upper arm occurring during parturition, which was first described by Duchenne. This combination does not, however, always occur under such circumstances; a case of this kind has been reported by Bailly and Onimus, and another has come under my observation, the following being a short abstract of the history:

Case IX.—Julius H., æt. 3 months, in robust health; the head was delivered by forceps, and the shoulders were then found to be tightly wedged in the pelvis, so that the accoucheur had great difficulty in disengaging them, and exerted considerable force in this manipulation. Immediately after delivery it was noticed that the left arm hung limp by the side, and the only movement observed in the limb was the power of flexion of the fingers. The measurements of the arms are exactly the same, and the child is so plump that it is impossible to tell whether the muscles are atrophied. When the arm is raised to the horizontal it drops down perfectly limp; irritation of the limb gives rise to no movements except flexion of the fingers. Examination shows that there is no fracture or dislocation of the humerus. None of the muscles, with the exception of the flexors of the fingers, react to the faradic current, and only slight contractions are observed on the application of the interrupted galvanic current; sensation appears to be normal.

In comparatively rare cases the entire limb is paralyzed as the result of a lesion to the peripheral nerves, usually from tumors growing from the brachial plexus, from the pressure of the dislocated head of the humerus, or from direct injury received during falls, etc. When the paralysis of the arm is complete, the prognosis is very gloomy, and complete recovery rarely, if ever, occurs.

Two cases of paralysis of the entire plexus are under my observation at present, one of which is due to a fall upon the shoulder, the exciting cause of the other being unknown. In the former, the circumflex and musculo-spinal nerves are chiefly affected (the degeneration-reaction is present in the corresponding muscles), in the latter the median and ulnar nerves are most seriously implicated.

---

[1] Berl. klin. Wschrft. No. 9, 1877. [2] Arch. f. Psych. IX. p. 738.

CHAPTER XII.

PARALYSIS OF THE NERVES OF THE LOWER LIMBS.

Paralysis of the Obturator Nerve.

Clinical History.

This nerve supplies the adductors of the thigh, the obturator muscles, and the gracilis and pectineus; it is also distributed to the integument of the inner aspect of the thigh in its lower two-thirds. This form of paralysis is extremely rare and its symptoms are not very marked; they consist merely of loss of the power of adduction of the thigh (adductors, gracilis, pectineus) and to a certain extent of external rotation (obturators). The paralysis may develop either on one or both sides, and occurs more frequently in combination with crural paralysis than separately. The loss of power is rarely very great and does not cause the patient very much inconvenience.

Etiology.

This form of paralysis may occur as the result of tumor growths upon the cauda equina, wounds of the lower portion of the spinal cord, psoas abscesses, pressure upon the nerve during forceps delivery or by a large fœtal head, wounds of the nerve after its exit from the pelvis. In one unique case, the notes of which were furnished me by Dr. V. P. Gibney, the paralysis was apparently due to over-exertion of the muscles, the result of excessive coitus.

Case X.—"Minnie M., æt. 19 years, a prostitute. Two years ago, the patient, according to her own account, had a vaginal abscess on the left side. Last July another abscess developed in the same locality, and this was opened. Upon attempting to get out of bed she found herself unable to walk and suffered from pains in the lower limbs; there were no cincture pains or d sturbance of the functions of the bladder. Two months elapsed before she began to go about, and she then could only walk very slowly; she has been gradually getting better. She now walks pretty well on a level, but gets upstairs with great difficulty; she is unable to adduct the thighs and carry one across the other.

"November 29th. The vagina was examined quite carefully (though the light was not good) but no cicatrix of an abscess could be found; the uterus appears to be in the normal position; no evidences of specific disease.

"The adductors of the thigh are found to be very feeble, especially in the right limb; these muscles are also very flabby; there is also some paresis of the extensors of the thigh. The faradic excitability is diminished in the adductor group of the right limb. The patient suffers from

nerves of the lower limb, on account of its great length and more exposed situation. It is comparatively infrequent, however, when compared with paralysis of the nerves of the upper limb. It may be due to hemorrhages within the spinal canal, to the pressure of tumors upon the cauda equina, to fracture of the lower lumbar vertebræ (in one case under my observation both sciatics were completely paralyzed in consequence of fracture of the second lumbar vertebræ with forward dislocation); it may also be caused by the pressure of intra-pelvic tumors, of the forceps or a large head during a severe and protracted delivery. After the exit of the nerve from the pelvis, traumatism plays a prominent part in the production of peripheral paralysis, and the nerve is usually involved after its bifurcation into the internal and external popliteal, the latter being most frequently affected. The injury may consist of a bullet or knife wound, blow with a club, a fall upon the buttocks, the pressure of a strait-jacket or, as in one interesting case which I observed, kneeling upon a ridged tin roof, which caused paralysis of the muscles supplied by the anterior tibial nerve; paralysis of the nerve after its exit from the pelvis may also be due to rheumatic influences (exposure to wet or cold). In rare cases, lead palsy or paralysis after acute infectious diseases may involve the muscles supplied by the sciatic, the former being restricted almost exclusively to the distribution of the anterior tibial nerve (extensors of the foot). Finally, a few cases have been reported in which paralysis occurred in the distribution of the sciatic nerve as the result of version by the foot during transverse presentation. I once saw a case of this kind in consultation, in which the paralysis was bilateral, and in which the accoucheur confessed that he had used great violence during version. In this case, however, the other nerves of the lower limbs were also paralyzed, and it seemed to me probable that the lesion consisted of a hemorrhage around the cauda equina.

Paralysis of the branches distributed to the biceps, semi-membranosus and semi-tendinosus, causes loss of the power of flexion of the leg upon the thigh. When the biceps contracts alone, it produces slight rotation of the leg outward; contraction of the semi-membranosus causes slight rotation inward. These movements are also lost in paralysis of the nerve above its bifurcation.

Paralysis of the internal popliteal gives rise to well-marked symptoms. Paralysis of the gastrocnemius, soleus, and plantaris causes loss of the power of raising the heel from the floor in walking, and therefore interferes very materially with locomotion. In severe forms contracture of the opposing extensor muscles of the foot develops, and gives rise to the production of talipes calcaneus. After this condition has continued for a long time, changes occur in the joint surfaces, but the consideration of these symptoms belongs to the orthopædic surgeon. Paralysis of the tibialis posticus interferes with extension of the tarsus upon the leg, inversion of the sole of the foot, and adduction of the foot. This gives rise to the production of calcaneo-valgus. Paralysis of the flexor longus digitorum and flexor longus pollicis causes loss of the power of flexion of the second and third phalanges. When the small muscles of the sole of the foot are paralyzed, their function is undoubtedly lost, but we possess such little voluntary power over the individual muscles, and the latter are so much engaged in the performance of reflex acts, that it is difficult and in many cases impossible to differentiate the action of one from the other. The muscles supplied by the internal popliteal are almost invariably paralyzed together.

Paralysis of the external popliteal is much more frequent than that of the internal popliteal or of the trunk of the nerve, as it is more exposed on account of its position in the anterior portion of the leg. Paralysis of the tibialis anticus interferes with flexion of the foot and with adduction to a certain extent, and also allows the antero-posterior arch of the foot to sink. When contracture of the antagonists occurs, therefore, pes equinus is produced. Paralysis of the extensor longus digitorum, extensor pollicis proprius and peroneus tertius also interferes with flexion of the foot, and at the same time, with abduction; contracture of the antagonists therefore leads to pes equino-varus. The peronei longus and brevis are extensors of the foot upon the leg, acting in combination with

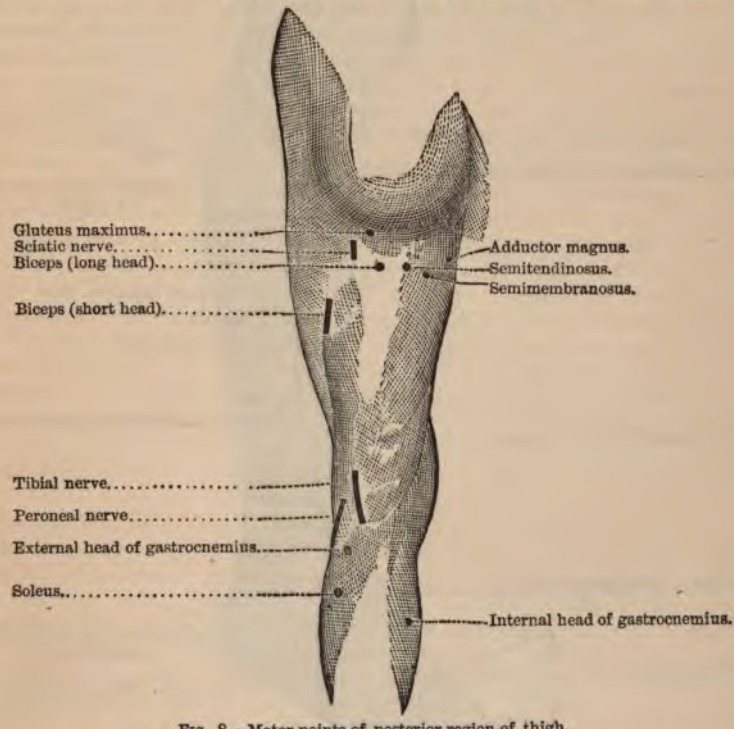


FIG. 8.—Motor points of posterior region of thigh.

the tibialis posticus in this respect. The peroneus longus also abducts the foot, and, at the same time, everts it. In addition, this muscle, as Duchenne has shown, holds the transverse arch of the sole of the foot in place by pressing the base of the first metatarsal bone against the adjacent bones. Its paralysis therefore gives rise to a certain degree of flat foot, which becomes complete if the tibialis anticus is also paralyzed.

DIAGNOSIS.

In these cases, also, it is often difficult to differentiate peripheral paralysis from infantile paralysis limited usually to the group of muscles supplied by the external popliteal nerve. It must be remembered that the analogous disease may also occur occasionally in the adult, and a case

of this kind has come under my notice in which it was difficult to exclude the peripheral character of the affection. The differential symptoms have been so often mentioned, that it is unnecessary to refer to them again.

If the entire nerve is paralyzed, and especially if the other nerves of the limb are implicated, we should make a careful examination of the pelvic viscera and also of the spinal column, in order to determine whether the paralysis may not be due to an affection of these organs.

We should also be on our guard against mistaking contracture of the muscles for paralysis, as such an error may be fraught with serious con-

FIG. 9.—Motor points of outer side of leg.

sequences in regard to treatment. A mistake of this nature can only be obviated by a careful examination of the affected parts, by observing which muscles are atrophied, and especially by noting the mobility of the parts. When the joints are very tender, it may become necessary to place the patient under ether in order to make a careful examination.

TREATMENT.

When the paralysis has been of long standing, and is complicated with contracture of unparalyzed muscles and with the various forms of talipes,

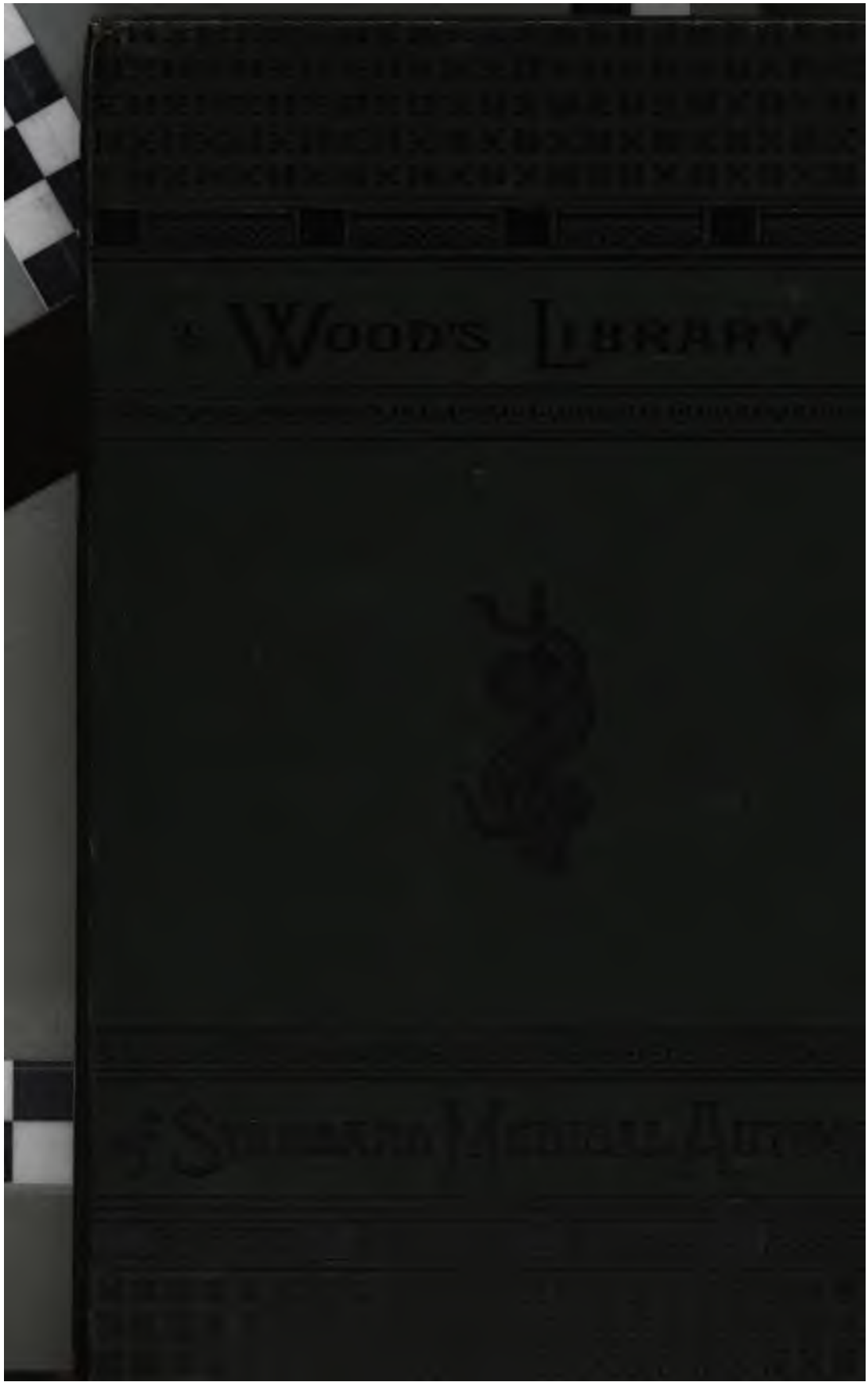
the treatment belongs properly in the hands of the orthopædic surgeon. The medical treatment consists entirely of the application of electricity, the current being varied according to the rules so often laid down. One electrode should be placed over the nerve, the other passed along the muscles. In the thigh, the nerve is readily found below the gluteal fold a little to the outside of the median line of the limb (Fig. 8); in the leg the external peroneal is found to the outside of the popliteal space, whence it winds over the head of the fibula; the internal popliteal is a little to the inside of the former (Fig. 9). The motor points of the muscles are very well shown upon these figures and need no further explanation.

In paralysis of the anterior group of muscles, considerable improvement in the power of walking may be obtained immediately by the application of an artificial muscle, in the manner employed by Dr. Sayre in the treatment of club-foot.

INDEX.

INDEX.


L341 Putzel,L. 928
P99 Treatise on common
1880 forms of functional ner
vous diseases


WOOD'S LIBRARY